VOLUME LXVII OCTOBER, 1957 NUMBER 10

THE LARYNGOSCOPE

FOUNDED IN 1890
BY
MAX A. GOLDSTEIN, M.D.

PUBLISHED BY
THE LARYNGOSCOPE
640 SOUTH KINGSHIGHWAY ST. LOUIS (10), MO., U.S.A.

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ST. LOUIS 10, MO., U. S. A.

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VOL. LXVII OCTOBER, 1957 No. 10

THE MECHANISM OF THE LARYNX.*

V. NEGUS, M.S.,

London, England.

To commence studying the relation of anatomical structure to function from the human aspect is to create confusion; man has passed through many phases in his evolutionary history and has emerged eventually as a versatile being, with no particular specialization of the body, but with a highly developed intellect. Then again man is a bad subject for study, because he belongs to the order of primates, a group of animals labelled as the highest of all, but actually a timid and defenseless collection which has taken to the trees in an attempt at escape from terrestrial carnivora, thereby undergoing considerable structural change.

An arboreal existence gave them some security and a new source of food, but even in the trees they are pursued by predatory enemies such as leopards and snakes.

For various reasons, therefore, it is proposed first to examine simple vertebrates, in which the complications of evolutionary progress have not advanced so far as to create confusion.

EVOLUTION OF THE LARYNX.

To seek a new source of food various aquatic animals have, in the course of evolution, ventured on to the land. An example, relevant to the present discussion, is the climbing

*Presented as the Samuel J. Crowe Memorial Lecture, Johns Hopkins Hospital, Baltimore, Md., May 2, 1957.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication May 15, 1957.

perch (anabas scandens). This fish has a diverticulum of specialized epithelium above the gills, capable of absorbing oxygen, but only if kept moist. This entails return to the water after a brief sojourn on land.

In other, more specialized fish, there is the desirability for breathing air at certain seasons of the year, when the water in which they live dries up in times of drought. These fish would either need to change their habitat, or alternately they would perish. The problem has been solved in these lung fish, or dipnoi, by the development of an air breathing apparatus, in the form of a lung; but a pulmonary outgrowth of an efficient pattern must be protected against inundation by water and this protection is afforded by a sphincter at the mouth of the pulmonary outgrowth, the musculature being derived from the floor of the pharynx. This specialized laryngeal girdle is of simple nature, but is efficient in closing the entrance to the lung, when water enters the pharynx. When the fish is out of water, and needs to breathe air, the sphincter relaxes and allows mouthfuls of air to be forced into the lung by means of a swallowing process.

MODIFICATIONS FOR RESPIRATION.

As evolution proceeds, and air breathing becomes habitual in terrestrial animals, there arises the desirability of active opening of this protective valve, in place of the previous passive relaxation of the muscle band. For this purpose, other fibres, also derived from the floor of the pharynx, assume a dilator function, to assist in pulling open the slit-like aperture; and to aid in the efficiency of this action, cartilaginous rods are developed in the margins of the larynx, to which the dilator fibres become attached.

One sees a further stage of evolutionary progress in the division of these lateral cartilages into separate segments, part appearing as the arytenoid cartilage and the remainder fusing with its fellow of the opposite side to form a cartilaginous ring, at first single, but later subdivided into cricoid and thyroid elements. To the arytenoid and to this fused ring, the sphincteric and dilator fibres gain attachment, with added

efficiency and with the production of a larynx of the type seen in amphibians, reptiles and birds.

The arytenoid cartilages in reptiles and birds are lengthy, extending along the entire margin of the glottis. They are hinged posteriorly and open as a triangle with the base in the anterior position. Such long cartilages do not vibrate readily and cannot produce anything more than a hiss when phonation is attempted.

Mammals show various changes in the size and shape of the arytenoid cartilages and in the attachment of muscles, but the basic principles of structure remain unchanged.

Opening of the Glottis.

The dilator muscles have already been mentioned as of later origin than the sphincteric elements. Arising as fibres derived from the pharyngeal musculature, attached originally to the margins of the glottis, but later with insertion into lateral cartilaginous bars, the dilator or abductor muscles of man work at a mechanical advantage by virtue of their backward migration, with their points of action on the muscular processes and posterior surfaces of the arytenoid cartilages, (see Fig. 1), thereby creating a greater effect with less muscular movement than in primitive species. By their action, the posterior muscles rotate the glottic processes of the arytenoids outwards and thus separate the vocal folds; but unlike the mechanism of many animals, a further action is executed by outward sliding of the arytenoids on their lax, inclined joints, under pull of laterally placed fibres of the posticus muscle. The glottis is thereby opened in a triangular manner, with its base placed posteriorly; in this, man is unlike most other mammals, in which rotation of the arytenoids gives to the glottis a quadrilateral or diamond shape and in birds a triangular shape, with the apex placed posteriorly.

In man the arytenoids are short in relation to the anteroposterior diameter of the glottis, the remainder of the boundaries being made up of the membranous inferior thyroarytenoid folds, usually known as vocal cords or vocal folds. Animals which require rapid and efficient respiratory change possess long arytenoids, the optimum length being seven-

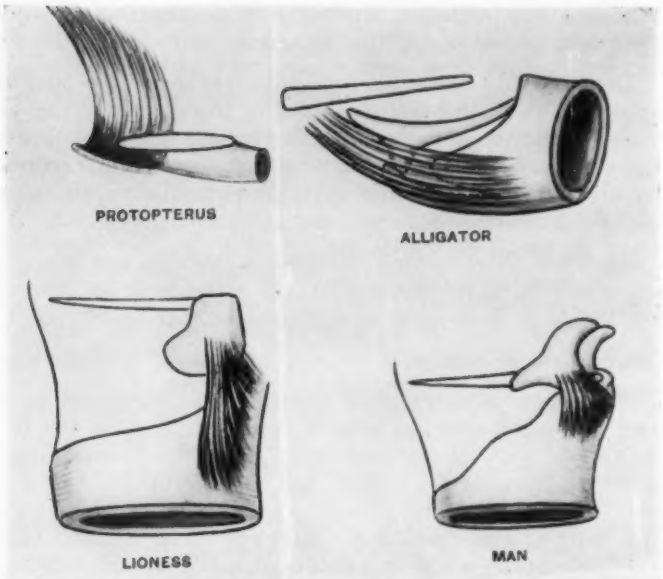


Fig. 1. Alteration in attachment of dilator muscles. In protopterus, the African lung fish, some circular muscle fibres in the floor of the pharynx terminate in the margin of the glottis at its anterior end. The alligator has dilator fibres arising from the crico-thyroid ring, inserted partly into the tip of the long arytenoid cartilage and partly into the margin of the glottis. The lioness (panthera leo) has a fan-shaped dilator muscle capable of rotating the short arytenoid cartilage outwards, thus opening the glottis. In man the muscle not only rotates the arytenoid cartilage but also slides it outwards.

tenths of the antero-posterior diameter of the larynx (see Fig. 2).

The shortness of the arytenoids of man is of benefit during swallowing, since the diameter of the laryngeal aperture can, in consequence, be considerably reduced when the phincteric muscles contract; rotation of the cricoid on the thyroid still further facilitates his shortening, thus permitting a bolus of food to pass readily into the mouth of the esophagus, which opens in a funnel-like manner to receive it.

The decrease in length of the arytenoid cartilages permits the membranous folds to be so much the longer, with advantages in phonation and fixation of the thorax.

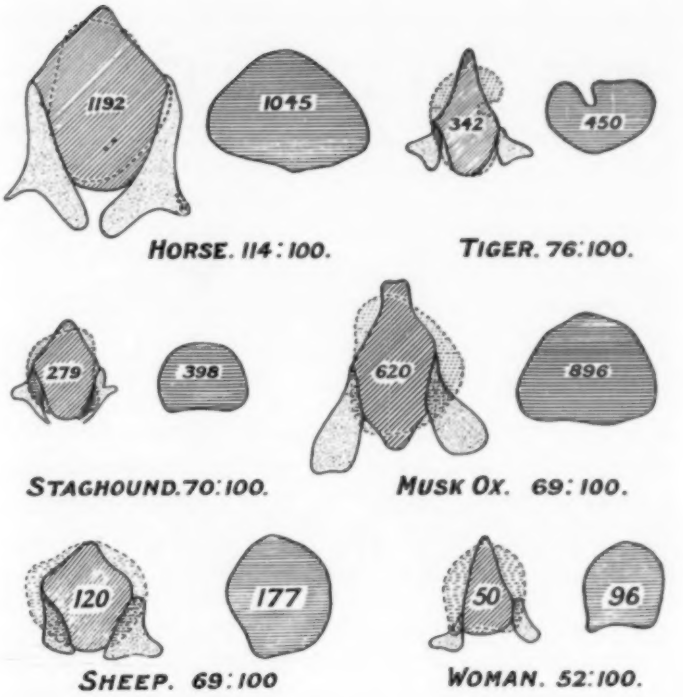


Fig. 2. Transverse section at the glottis and upper end of trachea. The outline of the section of the trachea is in each case superimposed on that of the glottis. The figures of sheep and woman are enlarged twice in comparison with the remainder. The ratio of cross sectional areas of glottis are given.

It must be observed that choking of the airway at the larynx is not necessarily an accompaniment of shortening of the arytenoids. If a more capacious glottis were required, it could be provided by increase in size of the larynx as a whole in the antero-posterior direction, as seen in many animals, such as members of the dog family. It is of interest to note that this relative increase is present in the human foetus and in infants, in whom the posterior plate of the cricoid cartilage has a backward inclination, thus giving to the larynx somewhat of a funnel shape and bringing its

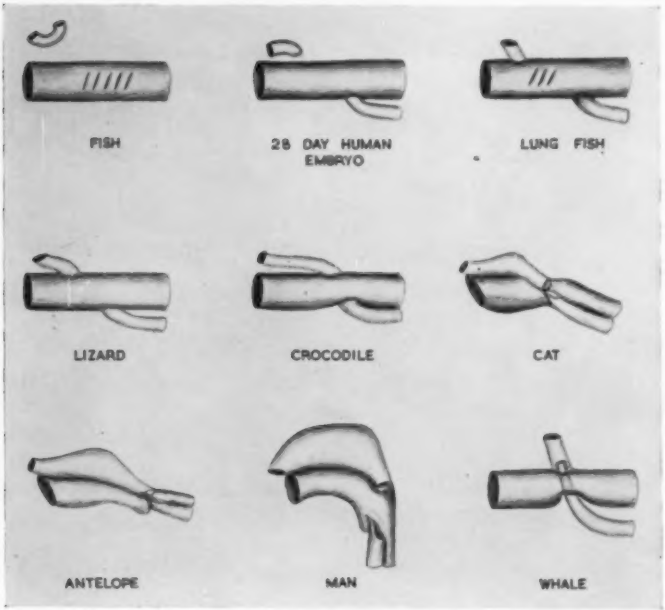


Fig. 3. Modifications to subserve olfaction. There is no communication between the nasal olfactory organ and the food tract in fish, with the exception of dipnoi or lung fish, or in the early embryo; in air-breathing species the two channels communicate. In most mammals there is coaptation of the nasal passages and the larynx, the intervening gap being closed by the epiglottis; the close relationship in keen scented cats and ungulates and also in cetaceans is illustrated. In man the larynx lies at a low level, with a wide gap between it and the palate.

cross-sectional area nearer to that of the trachea. In the male at puberty a similar increase is obtained, by growth changes affecting the antero-posterior diameter.

Direction of the Air Stream.

The primitive larynx may be described as a slit-like aperture lying flat in the floor of the pharynx; its opening is at a right angle to the axis of the trachea, with consequent friction (see Fig. 3). In the subseqent stages of evolution, with improvement in the rapidity of respiratory exchanges, it is found that a change in inclination occurs. The aperture of the larynx becomes tilted in such a way that the glottis

lies almost at right angles to the trachea. In man, with his relatively high aryepiglottic folds, projecting above the floor of the pharynx, the laryngeal aperture inclines somewhat backwards and is, in fact, thus directed so as to fall into line with the current of air arriving from the nasopharynx.

There are further points of importance to be considered in connection with the adaptation of the larynx for respiration, but certain of those related to the function of olfaction must first be considered.

ADAPTATIONS TO SERVE OLFACTION.

It is essential, if the olfactory sense is to be maintained at a fully efficient level, that air should pass over the olfactory nerve endings in the nose, even when the mouth is open. To this end, an epiglottis makes its appearance, and at the same time backward elongation of the soft palate is developed. The result of apposition between the long palate and the projecting epiglottis is the shuting off of the nasal and pharyngeal airways from the food passages, with conseqent elimination of mouth breathing when the mouth is open; scent-laden air is not diverted from the olfactory organ, as it would be if no such control of the air current were established (see Fig. 4).

Maintenance of the sense of smell at a high degree of acuity is of vital importance for the survival of the individual, not only in seeking and selecting food, whether animal or vegetable, but also in avoiding enemies.

In most keen-scented mammals the epiglottis assumes an intranarial position, lying on the upper surface of the soft palate; but in the dog it lies below the palate, but still with the desired result of obligatory inspiration through the nose.

In man, in contradistinction, vision is relied upon, and the acuity of the olfactory sense is of little importance and but poorly developed; in him there is no longer apposition of the epiglottis and soft palate. This results partly from lack of necessity, but mainly because of downward descent of the larynx, consequent on the assumption of the erect posture, with changes in the vertebro-occipital and pituitary

angles, and even more through recession of the jaws, which have no longer the prognathous character of most animals and of early man.

Intermediate stages between the intranarial epiglottis and that of man are seen in the higher apes, whose sense of smell is feeble, but whose jaws protrude and whose posture is slouching and not truly erect.

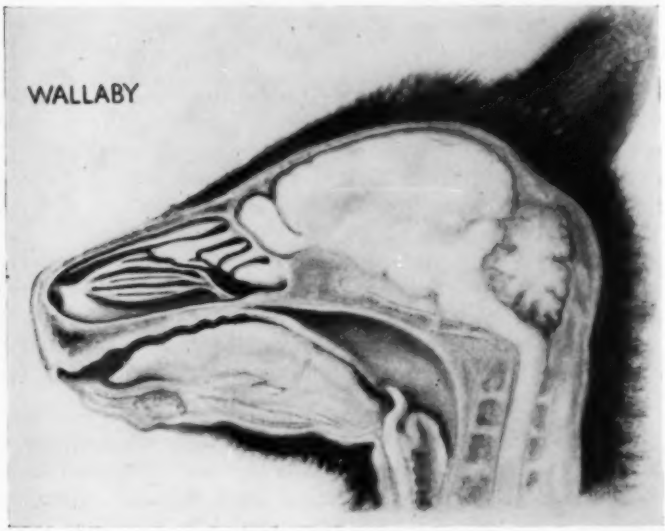


Fig. 4. Sagittal section of nose of a wallaby (macropus wallabatus). The anterior half of the nasal fossa is occupied by a grooved maxillo-turbinal body of wide area, designed for warming and moistening air; the posterior half lodges the olfactory ethmo-turbinal system. There is a large epiglottis lying above the soft palate, thus insuring nasal respiration when the mouth is open.

ACCESSORIES TO UNOBSTRUCTED RESPIRATION.

The tilting of the larynx to come into line with the nasal passages has been referred to.

To ensure continuity, and to eliminate points of friction as far as possible, the epiglottis plays its part. Evolved, as already described, for olfactory purposes, the epiglottis of

most mammals is also important in respiration, by closing the gap between the soft palate and the larynx; but in man the gap between the latter and the soft palate leaves an awkward hiatus; eddies arise and friction reduces the efficiency attained in other species with a continuous airway. What is lost in olfaction and respiration is gained in phonation, for man, with lack of apposition of palate and larynx, is enabled to emit sounds readily through the mouth, with advantages to speech.

Not only is there tilting of the larynx and continuity with the nasopharynx, but there is also straightening of the upper air passages in fast running animals, with consequent elimination of eddies and points of friction; these changes affect the anterior nares, the nasal fossae and the nasopharynx.

Man relies upon his intelligence for survival, rather than on his physical capabilities. He is neither so strong as the gorilla, orang or chimpanzee, nor so fleet as the cheetah, the gazelle or the horse. A man can run at about 20 miles an hour, a gazelle at 60 and a cheetah at 70 for short distances; and yet man has an ascendancy over these animals.

For reasons to be given later, obstruction at the nose and larynx is put to advantage for certain purposes of respiratory exchange, but always with an accompanying disadvantage of limitation of exertion. Obstruction at the nose can, to a certain extent, be counteracted by breathing through both nose and mouth; and this route is adopted by a runner, to his advantage, not only with increased respiratory exchange, but also with increased cooling of air, since the warming effect of the nose is correspondingly reduced.

Tortuosity of the air passages can be diminished slightly by extension of the head on the spinal column, as may be noted in a sprinter; but under no circumstances can the air tract be straight, as it is in most fast-running animals.

It is observed, therefore, that man, being versatile and with high development of certain faculties, suffers the disadvantages of a somewhat inefficient respiratory apparatus without reduction of his general superiority; for the loss in one direction is compensated by a gain in others.

The glottis of man is a choked point, its calibre being less than that of the trachea; obstruction to entrance or exit of air is, however, of advantage under certain conditions, to be discussed now.

RESPIRATORY MOVEMENTS AT THE GLOTTIS.

The choking at the glottis is of disadvantage only during severe exertion; at times of gentle exercise, or during resting periods, the aperture offers no undue obstruction. There are to be seen at the glottis movements of dilatation and constriction during respiration, and their purpose must be considered.

The anterior nares delay incoming air, measurements of air pressures in the nasopharynx showing a fall below that of the atmosphere during inspiration, and a rise on expiration; but there is a still further rise and fall in the trachea, by virtue of obstruction at the glottis. Observations show a widening of the glottis at the immediate commencement of respiration, as the result of active muscular contraction, more noticeable during exertion than at times of repose; and correspondingly, a narrowing during expiration, again sudden and active in its execution and slightly in advance of the movements of the chest wall. The reason for these rhythmic movements appears to depend upon the necessities of respiratory exchanges in the lungs; oxygen must be taken up by the blood from the air in the alveoli, and carbon dioxide must be given off, and these exchanges must be carried out rapidly, during the passage of blood through the pulmonary capillaries.

Pump Action.

The larynx exercises two functions: the first is directed to assist the circulation by a pump action. Partial obstruction at the glottis during inspiration assists in lowering intrathoracic pressure, and this in turn reduces pressure on the great veins and right auricle. A suction action is thereby brought into play, with attraction of blood from the systemic circulation into the right side of the heart. The same reduction of pressure leads to dilatation of the pulmonary

capillaries, with reduction of obstruction to the flow of blood through the lungs; in consequence more blood reaches the left auricle, and according to the law of the heart, an increased volume leaves the left ventricle, with a rise of aortic pressure.

Regulation of Respiratory Exchanges.

It is thus possible for the size of the glottis to correlate the amount of inspired air and the quantity of blood in the lungs to the best advantage, and this seems to explain the movement of glottic widening seen at the beginning of inspiration. During expiration, when air overcharged with carbon dioxide and deficient in oxygen is present in the lungs, the efficiency of respiratory exchange is much diminished; it leads to conservation of energy, therefore, if the flow of blood through the collapsing lungs is reduced during expiration. This is attained by increase of pressure in the thorax, and this again depends to a certain extent upon the obstruction at the glottis. If, therefore, there is partial closure of the vocal folds at the beginning of expiration, with slowing down of the expiratory air stream, there will be a corresponding slowing of the intra-pulmonary blood flow, the two being integrated according to the necessities of respiration (see Fig. 5).

Elimination of Dead Space Air.

Particularly in animals with a long trachea, reduction of the dead space during expiration is an important function, but one in which the larynx plays no part; the onus falls on the bronchioles, bronchi, trachea and nose.

Function of the Saccules.

Many animals have air sacs in different positions and of varying size, in connection with the respiratory tract; their purpose appears to be rebreathing of air, whereby there is mixing of air in the lungs with that in the bronchi, trachea, or nose; this mechanism is seen at its highest development in birds. In them, the lungs are small and almost immobile, but a wide series of air sacs is present; the air is caused to circulate to and fro through the lungs. In some of the

higher apes the laryngeal saccule is highly developed, and is continuous with large air sacs, which reach, in certain species, even into the axilla, for the purpose of rebreathing. In man the saccule is a small upward extension of the laryngeal ventricle and is of insignificant size, with no useful function either in respiration or phonation.

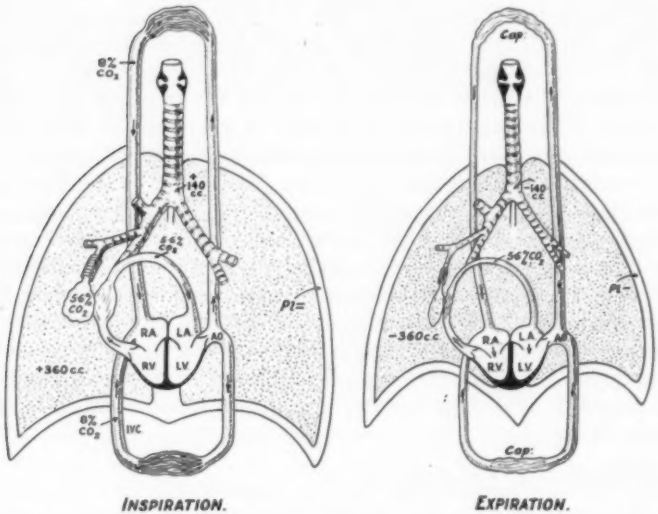


Fig. 5. Diagram to illustrate the dilatation of the pulmonary capillaries during inspiration and their collapse during expiration. The partial opening and closure of the glottis in the two phases is illustrated. The percentage of CO_2 in the venous and arterial systems and in the alveoli of the lung is shown. Cap=peripheral capillaries. RA=Right auricle. PL=Pleura. LA=Left auricle. RV, LV=Right and left ventricles. IVC=Inferior vena cava. The relative capacity of the lungs and the tracheobronchial tree is shown.

DEGLUTITION.

In animals which do not masticate, but bolt their food whole, the passage of food is rapid, and can be carried out during temporary cessation of respiration. This observation is not universally true, as, for instance, in the case of many snakes, which swallow such an enormous bolus—sometimes even a whole goat or pig—as to require some specialized

mechanism; but in carnivorous mammals, it may be taken as a general rule that the larynx is closed during deglutition, without inconvenience. Man, however, although partly carnivorous, is not completely so, and as his evolutionary history shows, his progenitors have passed through a herbivorous stage; his larynx shows evidence of this ancestry, and he gains advantage from the evolutionary changes so derived.

Swallowing of Liquids.

Animals which live on grass, herbage or leaves must swallow very large quantities of food to derive sufficient nourishment. Deglutition consequently occupies much time; and if respiration had to be interrupted during every movement of deglutition, there would be derangement of the respiratory mechanism. A means has been found, accordingly, to enable liquid or semi-liquid food to pass through the pharynx into the oesophagus, without closure of the larynx and without interruption of respiration. This end is attained by elevation of the margins of the larynx, whereby a lateral food channel is established, along which fluids may travel without inundation of the larynx and trachea. This protective boundary is the aryepiglottic fold, a barrier formed of a mucous membrane covering, with underlying submucous tissues and some contained muscle fibres; the fold stretches from the lateral margin of the epiglottis to the body of the arytenoid cartilage and its superimposed cartilage of Santorini. To assist its protective nature, there is present, in the aryepiglottic fold of many herbivorous animals and also in man, a supporting prop, the cartilage of Wrisberg (see Fig. 6). Braced back from behind by tonic contraction of the cricoarytenoideus posticus, the fold provides the medial wall of the lateral food channel. In man it is possible for saliva, or small quantities of swallowed fluids, to pass into the oesophagus even when the laryngeal aperture is open; in infants this function is even better developed, with considerable advantage, in view of their purely fluid nourishment.

Swallowing of Solids.

In herbivorous animals as well as in carnivora the larynx must be closed when a large bolus passes downwards. Clos-

ure of the larynx is brought about by a somewhat complicated mechanism, executed partly by the intrinsic muscles of the larynx itself, and in part by the musculature of the pharynx and hyoid system.

During swallowing of a bolus of any considerable size, whether liquid or solid, there is an initial elevation of the

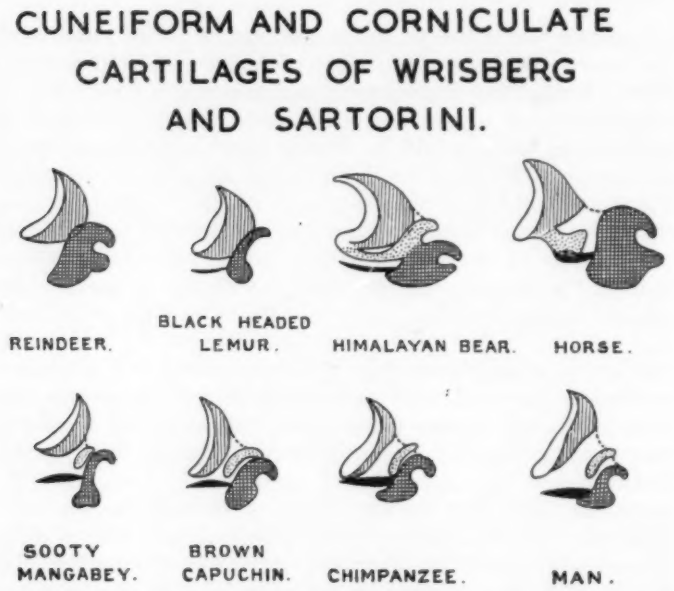


Fig. 6. In each case the epiglottic cartilage is lined vertically, the arytenoid is cross hatched, the cuneiform cartilage is stippled and the ventricle is darkly shaded.

pharynx, carried out by its longitudinal or vertical muscles. In this process of elevation, the larynx takes part, under the influence not only of the pharyngeal muscles, but also of other of the pretracheal group, notably the thyrohyoid. The larynx rises, not only upwards but also forwards, and is pulled by the muscles attached to the hyoid bone against the base of the tongue, which itself is markedly recurved during

transference of the bolus from the mouth into the pharynx; the thyroid cartilage slides up under cover of the hyoid bone. Being tilted forward, it follows that the body of the larynx compresses the epiglottis against the base of the tongue; the epiglottis is thus tilted backwards, and appears to turn down as a flap over the laryngeal aperture. It certainly assumes this position, but not with a purposive role; animals with no epiglottis, or human beings whose epiglottis has been removed by operation or disease, need suffer no disability.

Combined with these movements of the larynx as a whole, there is contraction of the intrinsic sphincteric muscles, and particularly of the fibres in the aryepiglottic folds, continuous below with the thyro-arytenoid muscle, and behind with the interarytenoideus. The latter, lying below the recurved cartilages of Santorini, tilts them forward and so brings the arytenoid cartilages forward towards the cushion of the epiglottis. At the same time, and by the same sphincteric action, the cricoid cartilage is tilted forward on its joints with the inferior cornua of the thyroid cartilage, thus assisting in diminution of the antero-posterior diameter of the larynx.

Closure of the laryngeal aperture, by an inward and forward infolding movement of the flexible aryepiglottic folds, thus takes place in a purse-string manner. To allow this to occur, there is relaxation of the posterior crico-arytenoid muscles, which normally brace back the aryepiglottic folds, and also of the cricothyroid muscles, which pull the thyroid and cricoid cartilages apart.

As the bolus approaches the hypopharynx, a sensory stimulus, initiated in the posterior pharyngeal wall, causes inhibition of the cricopharyngeal sphincter. Relaxation of this muscle not only gives freedom to the upward and forward movement of the larynx, but at the same time allows the mouth of the oesophagus to open in a funnel-like manner. This opening is assisted by drawing upward and forward the anterior wall of the oesophagus, part of which is attached to the recurved tips of the cartilages of Santorini. There is seen, in this mechanism, a very beautiful co-ordination whereby the larynx closes, rises and passes forward so as not to

impede the descending bolus while preventing entrance of food into the trachea; and at the same time the oesophagus opens widely to receive the swallowed mass. Lack of co-ordination of these complicated movements sometimes leads, in man, to the formation of a pharyngeal pouch.

REGULATION OF INTRATHORACIC AIR PRESSURE.

In addition to its important function of helping to control the air-stream as it enters and leaves the lungs, there is another aspect of the valvular glottis to be considered. This concerns complete interruption, either of inspiration or expiration, as a means of assisting fixation of the thorax.

Prevention of Entrance of Air.

The simple sphincteric larynx of the lung fish is not well designed to resist high degrees of alteration in air pressure, but in certain amphibians and reptiles, such a valvular type is present. In herbivorous types, elevation of the margins of the larynx, with the creation of a lateral food channel, leads to the formation of an organ which is of non-valvular nature in those species with a purely terrestrial habitat; but if the larynx of herbivorous animals with arboreal habits be examined, one will observe that valvular folds are present in the margins of the glottis. These folds are upturned and in many instances are of an efficient valvular type, somewhat similar to the aortic valves of the mammalian heart. It is obvious that these valvular folds, if brought into mutual apposition, will efficiently prevent air from entering the trachea. An examination of the larynx of all mammals shows that the possessors of this type have similar habits of feeding on berries, nuts or leaves, and of making independent prehensive use of the fore-limbs for grasping or climbing (see Fig. 7).

Man shows a similar structure, although to a somewhat less efficient degree; in him secondary inferior thyro-arytenoid folds have been evolved as fixation valves, to be used for a purpose similar to that of arboreal animals, as will shortly be explained.

When an animal hangs on to the branch of a tree, or pulls

itself up by the fore-limbs, or when it hugs—as in the case of a bear—or when a man lifts a heavy weight, the pectoral muscles play an important part. These muscles arise from the ribs, and are inserted into the humerus; as they contract they can either elevate the ribs, as in forced respiration, or alternatively they can depress or adduct the fore-limb. When the latter action is required, means must be found of immobilizing the ribs, if the muscles are to function efficiently. The ribs can be held down by muscular action, mainly by the muscles of the abdominal wall, but assistance can also be given through the intermediary of the larynx. If the glottis be closed at the beginning of the fore-limb effort so as to prevent air from entering the trachea, then any tendency to expansion of the thorax will be counteracted, and resistance will be offered to elevation of the ribs. It is, of course, necessary that during this action the diaphragm should contract, as in an ordinary inspiratory effort.

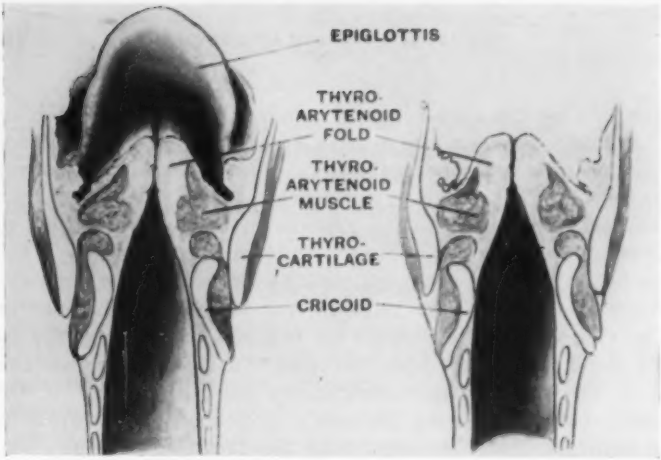
This, then, appears to be the primary function of the inferior thyro-arytenoid fold, such as that of lemurs, monkeys, apes and man. The fold is used also for phonation, and is, therefore, generally referred to as the vocal cord, even though its use for this purpose is a later and superadded function.

It is of interest to note that fixation of the thorax in this position of inspiration, with temporary reduction of intrathoracic pressure, is of physiological advantage, in that it assists in the pump action of the heart and facilitates the intrapulmonary circulation.

The ventricle comes into existence as a result of the formation of this secondary fixation valve; it is left as a recess lateral to the inferior thyro-arytenoid fold, lined by an epithelial membrane, beneath which is placed part of the thyro-arytenoid muscle.

Prevention of Escape of Air.

Under certain conditions the larynx assists in maintaining intrathoracic pressure at a level in excess of the normal. This occurs during straining movements, at times when pressure has to be exerted on the abdominal viscera, as in defaecation. Compression by the anterior abdominal wall would naturally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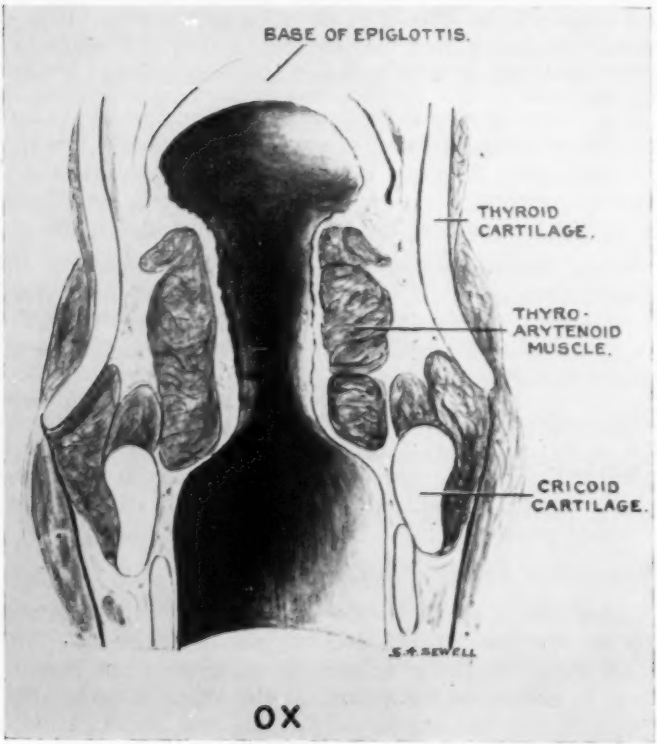
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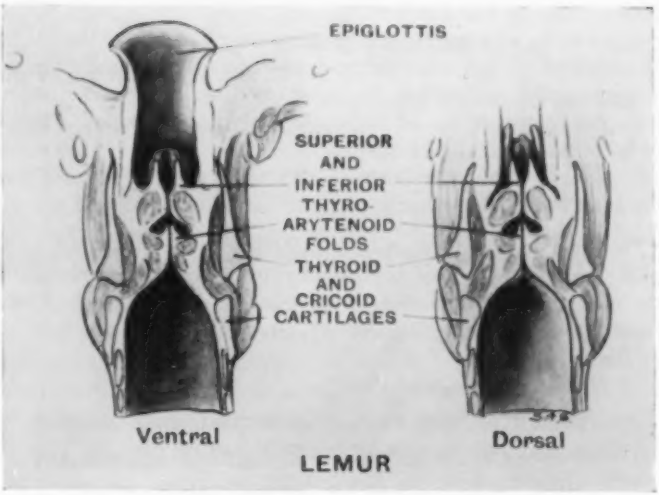

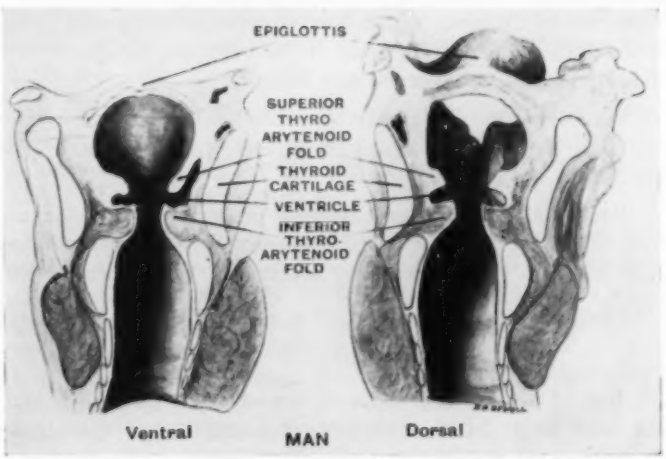

Fig. 7. Regulation of intra-thoracic pressure by the larynx. The ox (bos taurus) has a non-valvular glottis, closure being carried out by a powerful sphincteric girdle of muscle. The lion (panthera leo) has a primitive type of larynx with an undivided thyro-arytenoid fold and no ary-epiglottic fold. Man has a valvular glottis, with slightly upturned inferior and downturned superior thyro-arytenoid folds, both valves being capable of resisting pressure. The lemur has a very powerful inferior inlet valve and an upturned superior fold, both designed to resist entrance of air.

tend to elevate the diaphragm, and resistance to such movements can be assisted by the imprisonment of air in the lungs. To this end the superior thyro-arytenoid folds, usually known as ventricular bands, are brought together by contraction of the sphincteric group of intrinsic laryngeal muscles. These folds, with their down-turned margins, are able to resist very considerable pressure if the chest be filled and the thoracic walls compressed.

The mechanism is physiologically unsound, in so far as it leads to compression of the great veins, the right auricle and the pulmonary capillaries, with consequent slowing of the circulation and damming up of blood in the right side of the heart.

PROTECTION OF THE TRACHEOBRONCHIAL TREE AGAINST ENTRANCE OF FOREIGN BODIES.

The primitive function of the larynx was described earlier in this communication as protective, to exclude food or water. It carries on this function in higher types with closure either at the level of the glottis, or, in herbivorous species, above this level at the aperture of the larynx.

Even with effective means of exclusion, objects sometimes pass into the trachea and bronchi, being carried in by the inspiratory air stream.

Ciliary action is not an effective protector of the larynx, since there are no cilia on the inferior thyroid arytenoid or vocal folds; ciliary streams are confined to the interarytenoid region.

PHONATION.

It will be noticed that this subject has been left to the last, as a function not comparable in vital importance with those connected with respiration and deglutition.

Almost any larynx serves for the production of sound if air be blown through the glottis. A simple illustration is an artificial larynx constructed of two small strips of muscle from the thigh of a frog, pinned side by side over an aperture

connected with a pressure pump. If air be blown through, the strips vibrate and sound is produced, rising in pitch if the elasticity of the muscle be raised by Faradic stimulation; the sound is louder if a resonating chamber be applied to the preparation.

This experiment illustrates the mechanism of phonation at the larynx; the lungs supply the air pressure, the inferior thyro-arytenoid folds act as the vibrating reed, and the pharynx, mouth and nasal passages are the resonators.

The glottis of a reptile or bird is not well adapted for sound produuction, because of the long and rigid arytenoid cartilages; but the glottis of a cat, bounded by undivided thyro-arytenoid folds, is admirably suited for phonation and is made much use of.

The inferior divisions of the divided thyro-arytenoid folds of primates, with short arytenoid cartilages and long membranous folds, bounded by thyro-arytenoid muscles of variable elasticity, give to monkeys, apes and man an organ well suited to the production of sounds of wide pitch and satisfactory quality.

If air be blown through the excised larynx of a monkey or baboon, sound is produced, the pitch rising slightly on increase of air pressure if the degree of tension of the margins of the glottis be constant.

The thyro-arytenoid muscle, consisting of an internal and an external division, is the vital factor in phonation; its attachment to the body of the arytenoid cartilage enables the glottic margin to acquire elasticity, with opposition by the posterior crico-arytenoid muscle. Apposition of the arytenoid cartilages, essential during phonation, is assisted by a rotating action of the lateral crico-arytenoid, and by a movement of approximation by the interarytenoid muscle.

The other intrinsic muscles of the larynx act in unison with the thyro-arytenoids, to regulate the length of the glottis and the degree of elasticity, in relation to the pressure of air escaping from the trachea. The position of the larynx itself is regulated by the various muscles attached to it.

The glottic margins alternately separate when blown apart by the intra-tracheal pressure and recoil when the pressure is thereby reduced; rhythmical vibration produces sounds of fixed pitch, and these sounds are then amplified and modified by the resonators. Volume or intensity varies with the force of air pressure.

Certain overtones may be accentuated to give various patterns to the mixed laryngeal sounds, by virtue of alteration in shape and size of the superimposed resonators, namely the pharynx, mouth and nasal cavities; in this way the different vowel sounds can be distinguished by the ear. Quality is an attribute of the resonators and is not determined primarily by the larynx.

From the original involuntary sounds, later adapted to purposive cries and still further elaborated into vowels and consonants, language is built up. A large buccal cavity, bounded by cheeks evolved for mastication and closed by lips provided for suckling, confer considerable advantages. Freedom of escape of air through the mouth allows full use to be made of the buccal structures.

Man's intelligence has allowed him to take advantage of the laryngeal organ so as to elaborate a code of communication. This ability to convey messages and ideas to others has in its turn increased his intelligence and has placed him in a commanding position in the animal kingdom.

Reduction of the length of the arytenoid cartilages, with elongation of the membranous folds, separation of the epiglottis from the soft palate as the result of descent of the larynx and reliance on sight rather than on smell, and the provision of a valvular type of inferior thyro-arytenoid fold, have given to man a vocal apparatus of great efficiency. Regression of certain characteristics, such as speed in running, keenness of sense of smell and arboreal activity, have been compensated by general versatility.

Although examples have been given of very simple and easily controlled types of vocal apparatus, yet in the case of man, with his ability to produce tones of wide range and of good quality, the task imposed on the central and basal nerve

centers is one of great delicacy. Over 100 single or paired muscles, controlling the thorax and diaphragm, the larynx, the resonators and the vocal stops, must be adjusted accurately, with rapid changes in singing.

The question of registers arises in the human vocal range and further consideration must be given to the thyro-arytenoid muscles, of prime importance in this respect. On low pitches considerable broad surfaces of the vocal folds are in contact, with only slight contraction of the thyro-arytenoid; the large mass of muscle when blown apart recoils relatively slowly, with production of a low pitch. High speed moving pictures show the rolling apart of these broad folds, commencing below and finishing above, with escape of a puff of air.

Many fibres of the external thyro-arytenoid muscle arise in man, but not in animals, from the lower part of the conus elasticus, the upper margin of which forms the fibrous covering of the vocal folds; on higher pitches contraction of these fibres, as elasticity of the glottic margins is increased, pulls the lower part of the conus and vocal folds outward, and in consequence the areas in apposition are diminished. Instead of thick margins the glottis is then bounded by thinner folds, which can produce tones of high pitch. The two registers are known as thick and thin, or sometimes, incorrectly in my opinion, as head and chest registers.

Some observers are not satisfied with this tonic explanation of phonation, in which the active agent is raised intra-tracheal pressure, with passive separation and recoil of the elastic glottic margins. There has been propounded a clonic theory, which considers the muscles of the glottic margins to contract and relax under rhythmical nerve stimulation, at a rate corresponding to that of the pitch to be produced, and with passive escape of puffs of air from the trachea. The recording of action potentials in the glottic muscles is advanced in support of their views.

According to my interpretation of various communications, the authors concerned do not appear to dispute the validity of any part of the mechanism except that which concerns the thyro-arytenoid muscles.

The range of the human voice rises as high as 2,048 C/S, and the production of such a pitch appears to throw an impossible burden on the clonic theory.

It is further suggested by the advocates of this mechanism that the margins of the glottis are pulled aside by the oblique fibres inserted into the conus elasticus. Anatomically, opening of the glottis by this means would appear practically impossible in man and completely so in such an animal as a lemur or gibbon, with no such oblique fibres and with sharp edged vocal folds, in some instances resembling aortic cusps.

Professor Neil and Dr. Floyd, of the department of physiology of the Middlesex Hospital, London, have joined with me in the investigation of this problem and have applied their knowledge, experience and wealth of scientific apparatus to a solution of the problem.

In the first place it is on record by Ladd Prosser that the fastest rate of rhythmic contraction of skeletal muscle known is that recorded for the muscles of the wing of a fly, at a rate of about 330 per second.

Secondly, the mere demonstration of discrete action potentials in laryngeal muscles during stimulation, as reported by the exponents of the clonic theory, is no guarantee that the muscle is contracting rhythmically. Sherrington showed years ago that rhythmical stimulation of skeletal muscle results in fused or tetanic contraction, even at the relatively low rate of 67 stimuli a second; the electrical response of the muscle, however, shows the setting up of action potentials at 67 per second, even though the muscle is in tonic contraction.

Experiments were carried out in the Middlesex Hospital Medical School on the larynges of cats, with rhythmical stimulation of the recurrent laryngeal nerve; the electro myographic response, (e.m.g.) of the thyro-arytenoid muscle being recorded at the same time by means of a cathode ray oscilloscope. At frequencies of 50 and 100 C/S a large response followed each stimulus and at rates as high as 250 C/S occasional response was found in the muscle.

At higher frequencies, however, there was no obvious relationship between the e.m.g. potentials and the stimulation frequency.

These experiments indicate that although the muscles bounding the glottis might contract rhythmically at low rates, yet at higher frequencies they obey the laws of other muscles and go into tetanic or tonic contraction, as envisaged in the usual theory of phonation.

A second series of experiments was carried out by means of a Piezo electric transducer. This consists of a slender steel wire resting against the free margin of the thyro-arytenoid fold, its other end being fixed to a diaphragm which conveys vibrations to a crystal; compression of the crystal produces currents which can be recorded on an oscilloscope, thus giving a tracing of the actual mechanical movements of the vocal fold.

Rhythmical stimulation of the recurrent nerve of one side showed that a rate of 90 stimuli per second causes vibration of the muscle at the same rate; a similar result was recorded at 100 C/S, although at this rate the clonic contractions were much smaller in amplitude. At 110 shocks per second no clonic movements occurred.

To make certain that the apparatus was capable of recording higher rates of vibration, the tip of the transducer was placed in contact with the diaphragm of an earphone, which was made to vibrate at various frequencies.

The transducer was found to record accurately oscillations at rates of 250 and even 900 C/S.

The evidence of comparative anatomy, of mechanical experimentation and of investigation by various electrophysiological methods leads to the conclusion that the clonic theory is untenable.

CONCLUSION.

Comparative anatomy and physiology are of considerable value in interpreting the various functions of the larynx.

The primitive function can be explained and the various modifications designed to meet the many demands on the organ become intelligible.

Although often described as the organ of voice, yet the adoption of an already modified organ for this secondary purpose appears clear.

Especially when dealing with the production of sound, the point is emphasized that the larynx of man follows the general physiological principles applicable to other vertebrates and is not subject to mechanisms of purely human design.

ERRATA.

The following legends for the paper "Congenital Choanal Atresia" by Col. Byron G. McKibben, M.C., U. S. A., published in THE LARYNGOSCOPE August, 1957, were ommitted at time of publication.

Fig. 1. Diagrammatic sketches showing the embryological development of the nasal, oral and pharyngeal cavities, and the choanae. The pharyngeal membrane is absorbed after the fifteenth day of embryonic life[21] and the bucconasal membrane after the thirty-fifth day.[27a] (Redrawn from Colver.[25])

Fig. 2—Roof of primitive mouth, viewed from below, at successive stages in development of palate and primitive posterior nares. Pal. proc—palatal process. Prim. ant. naris—primitive anterior naris (choana). (Reprinted from Durward, et al.,[9] after Frazer.)

Fig. 3. Photomicrograph of a frontal section through the ventral portion of the nose of a human embryo, aged 49 days. Note the epithelial plugs occluding the nasal cavities. (By permission from The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man, by Schaeffer, J. Parsons, 1920, Fig. 14, p. 10, McGraw-Hill Book Company, Incorporated.)

Fig. 4. After a dissection of a newborn male infant with complete occlusion of both choanae. The atretic structure consisted of a thin fibrous sheet lying between the nasal and pharyngeal mucous membranes. (After Schaeffer and Riggle) Courtesy of Pancoast, H. K., Pendergrass, E. P., and Schaeffer, J. P.: The Head and Neck in Roentgen Diagnosis, Charles C. Thomas, Springfield, Ill., 1940, Fig. 539, p. 345.

Fig. 5. Diagram showing portion of nasal septum to be removed. The mucosal layers cover the cut edge of the bone and encourage more rapid healing. This is more easily accomplished through the transpalatine approach. B—edge of bone, M—edge of mucous membrane, P—posterior wall of nasopharynx, V—posterior border of vomer, removed.

SOME RELATIONS BETWEEN AUDITORY FUNCTION AND INTRACRANIAL LESIONS WITH PARTICULAR REFERENCE TO LESIONS OF THE CEREBELLOPONTINE ANGLE.*

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This report is a review of 18 cases on which neurosurgical or neurologic evidence is available: *1.* to see what general statements may be made at this time about auditory function in intracranial lesions, and *2.* to see what auditory signs may now be designated as indicative of intracranial lesions. The four cases presented in the 1955 report of Walsh and Goodman[1] are included in the present series.

In terms of diagnostic goals, proper evaluation of auditory function may, *1.* provide information corroborative of other neurologic observations where the diagnosis is fairly clear; *2.* suggest the site of the lesion in cases where the neurologic signs clearly indicate intracranial disturbance, but the location of the lesion is in doubt (Shephard and Wadia's report[2] on atypical features in acoustic neuroma underscores the role of auditory measures in this situation), and *3.* provide, for lesions of the cerebellopontine angle, in particular, an early diagnosis in the absence of extensive neurologic signs. That examination of auditory function may have a major role in early diagnosis follows from these general observations: *1.* In 70-80 per cent of lesions of the cerebellopontine angle reported, auditory symptoms precede all other symptoms by about 18 months on the average.[3,4,5,6,7,8] It is reasonable to assume, therefore, that the first clear signs are likely to be auditory. *2.* There is more to the measurement of auditory function than the conventional threshold audiogram.

These observations, the recent reports of Bocca[9] and his co-workers, and those of Goldstein, Goodman and King,[10]

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Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication July 29, 1957.

which relate a pattern of auditory dysfunction to lesions of the temporal lobe suggest that the problem of establishing the auditory concomitants of intracranial lesions can be reduced to two general areas of effort: *1.* The "isolation" of the finer complexities of auditory function, for example, pitch and loudness discriminations, response to prolonged stimuli, the discrimination of complex stimuli; *2.* the establishment of clear relations between more than one auditory function and certain loci of intracranial disturbance.

The present study is concerned with thresholds for pure tones and for speech, and to those above-threshold functions which relate to discrimination for speech and certain pitch and loudness discriminations. This limitation of the extent of examination reflects curtailment due to circumstance rather than restriction by preference. The evidence to be presented suggests that the relations between the threshold for pure tones and discrimination for speech, modified by the presence or absence of loudness recruitment, provide significant information in the clinical diagnosis of some intracranial lesions.

EQUIPMENT AND PROCEDURES.

Threshold for speech was measured with CID Auditory test W-2,[11] a set of English spondaic words whose intensity decreases automatically on the record. The words were presented to one ear at a time through earphones. The values given as hearing loss for speech represent the difference in decibels between the threshold for speech of the patient and the normal threshold for speech under the same testing conditions.

Discrimination for speech was measured with the Rush Hughes recordings of the PB monosyllable word lists (Rush Hughes PB-50 Auditory Test). These lists of relatively difficult words were presented to one ear at a time through earphones, at a level at least 40 db. above the patient's threshold for speech (unless such a level exceeded 120 db. SPL or was uncomfortable for the patient) since this high sensation level is sufficient to establish a maximum discrimination score.[12] A discussion of the diagnostic value of this set of recordings and of the problems involved in the use of other

versions of these word-lists is contained in a recent report by Silverman and Hirsh.[13] An additional measure of discrimination for speech was obtained in some instances with Auditory Test W-22, an "easier" version of the PB word test than the Rush Hughes recordings.[11,12]

In some cases the patients were unable to identify any of the monosyllabic or spondaic words. When this occurred, some indication of hearing for speech via the microphone and associated amplifiers and earphone was attempted. Spondaic words were presented at a level high enough to be heard comfortably and were very carefully enunciated. When this failed to yield a correct response, easier speech in the form of numbers or questions (usually the same questions used in the interview) were delivered to the ear under test. When the patient was unable to respond correctly to any of these speech signals (and the patient reported that the signal could be heard comfortably) the results of the speech-hearing examination were summarized as "no discrimination for any speech" or "total loss of discrimination for speech".

Details of the equipment and procedures used for the measurement of threshold for pure tones, for loudness balance and for diplacusis are reported elsewhere.[1,14]

In order to eliminate responses from the ear not under test when the threshold difference between ears was 35 db. or more, a white-noise masking signal was presented to the better ear at over-all levels of 80 to 95 db. SPL, depending upon the threshold difference, while measurements of threshold (tones and speech) and discrimination for speech were determined for the poorer ear. The opposite ear was masked in all measures of threshold by bone conduction.

PLAN OF THE STUDY.

Corresponding to the diagnostic distinctions that are usually required in the clinic there are three gross anatomic distinctions to be made: *1.* the auditory system central to the cochlea; *2.* the cochlea itself; *3.* the auditory system peripheral to the cochlea. In most instances the major difficulties in differential diagnosis lie in the separation of cochlear

disease from disturbance central to the cochlea. This distinction provides the primary question of the present study. The plan of this presentation, therefore, is to contrast auditory function in cochlear lesions with function observed in lesions central to the cochlea. The group of cases with cochlear disease serves in effect as a control group. This group must be defined for the most part on the basis of clinical rather than surgical observations. The attempt, therefore, has been to employ criteria for its designation which are as independent as possible from the auditory functions which serve as the variables with respect to which both groups are compared. There is evidence that Meniere's disease is directly related to cochlear dysfunction.[15] While this appears equally true of noise-induced hearing loss, among others, as a disease entity, Meniere's disease has been selected as representative of cochlear pathology as a matter of convenience. In this context, the auditory criterion for the designation of cochlear disease was a measured remission in hearing loss or discrimination loss for speech. Since hearing apparently does not fluctuate widely in other diseases affecting audition, the assumption is that when such remission is observed, the disease is in the category of labyrinthine hydrops, or Meniere's disease. Given negative otoscopic and general medical observations, whether the remission is spontaneous or following treatment is not material to the assumption.

RESULTS.

In the interest of ease of presentation of data, bone conduction measures are not shown in the tables of data. In all cases the thresholds for bone-conducted pure tones were grossly equal to the air-conduction thresholds.

Table I presents the complete audiometric data for the cochlear-disease group. Eight of the ten cases meet the primary criterion of a measured remission in hearing loss. The two remaining cases with similar symptoms (RE and VB) underwent surgical exploration of the cerebellopontine angle with negative findings.

Table II presents the complete audiometric data for the cases with intracranial lesions. Twelve are cases with lesions

TABLE I.

AUDIOMETRIC DATA FOR PATIENTS WITH COCHLEAR LESIONS.

			Hearing Loss Db.													
			Pure Tones, C.p.s., Air Conduction†								Speech	Discrimination Score		Speech-Hearing At Later Exam.		
Case	Age	Date	250	500	1000	2000	3000	4000	6000	8000	W-2*	RH**	W-22***	Date	W-2*	RH**
JR	49	4/56	35	35	30	35	30	35	45	55	33	51	76	6/56	17	86
EH	75	5/53	45	35	30	25	45	45	55	35	42	28	—	2/54	16	76
MB	33	11/53	40	45	55	50	30	35	45	50	58	30	—	1/54	27	54
JHB	27	10/51	55	45	55	35	40	35	40	45	41	44	—	3/52	12	94
WS	57	10/54	50	55	50	20	25	35	25	15	46	32	—	11/54	2	82
RE	44		40	45	60	60	65	65	70	70	56	30	72	(See text)		
WZ	60	2/54	50	55	60	65	65	65	80	65	81	2	—	2/54	27	48
CJ	39	11/55	45	60	65	55	50	65	80↓	80↓	85	6	—	2/56	15	64
BA	28	7/54	55	65	60	55	45	45	60	70	77	6	—	9/54	18	74
VB	45		65	70	75	60	90	90	80↓	80↓	92	0	—	(See text)		

†Bone conduction thresholds are grossly equal to air conduction thresholds.

*Spondaic words. Recorded version: CID Auditory Test W-2, descending intensity recording.

**Monosyllabic words. Recorded version: Rush Hughes recording, PB-50 Auditory Test. Values are per cent words correctly identified.

***Monosyllabic words. Recorded version: CID Auditory Test W-22. Ira Hirsh recording of modified PB-50 lists. Values are per cent correctly identified.

TABLE II.
AUDIOMETRIC DATA FOR PATIENTS WITH INTRACRANIAL LESIONS.

Case	Age	Date	Hearing Loss, Db. Pure Tones, C.p.s., Air Conduction† 250	500	1000	2000	3000	4000	6000	8000	Speech Discrimination Score W-2*	RH**	W-22***	Diagnosis
DB	38	4/53	5	5	0	15	50	55	55	60	4	44	—	
		11/56	40	35	40	95↓	95↓	95↓	80↓	80↓	38	0	0	Angle tumor, confirmed
GG#	37		0	5	20	20	35	35	15	20	7	56	—	Angle lesion, presumed
LR#	54	3/53	40	35	40	55	55	65	75	80	47	42	—	
		4/53	70	65	65	60	60	65	70	80	68	5	—	Angle lesion, confirmed
HG	65		30	45	40	45	65	65	65	65	43	67	—	Angle lesion, confirmed
MS	21	(left)	25	15	25	95↓	95↓	95↓	80↓	80↓	No Discrimination			Angle tumor, confirmed
		(right)	15	20	40	50	30	15	20	25	No Discrimination			Angle tumor, confirmed
		(right)	10	10	10	15	95↓	95↓	80↓	80↓	16	0	0	(Post-operative)
DW	35		10	45	60	50	40	40	30	25	70	0	—	Angle tumor, confirmed
JB	45		50	50	50	40	45	50	60	55	60	8	44	Angle lesion, confirmed
LB#	34		60	65	65	45	40	50	40	40	No Discrimination			Angle tumor, confirmed
DL	52	5/55	65	65	70	60	75	80	80↓	80↓	No Discrimination			
		2/56	65	70	70	60	75	80	80↓	80↓	96	0	0	Angle lesion, presumed
KF	62		75	70	85	90	75	95	80↓	80↓	No Discrimination			Angle lesion, presumed
DH#	54		80	85	80	75	80	80	80↓	80↓	No Discrimination			Angle tumor, confirmed
JJ	12	(right)	5	10	10	15	15	15	10	0	No Discrimination			Tumor, Third
		(left)	15	10	5	15	15	10	20	–5	No Discrimination			Ventricle, confirmed
JW	45		(highly unreliable; see text)								No Discrimination			Multiple tumors, presumed
TW##	30		10	5	5	0	–10	10	25	10	0	52	84	Left hemispherectomy
DS##	13		–10	–10	–10	–5	–5	–5	–5	–10	2	68	96	Left hemispherectomy
WM##	37		–10	–10	–10	0	10	15	0	10	8	54	82	Left hemispherectomy
EW##	37		–5	0	0	0	15	10	20	25	3	46	—	Left hemispherectomy

†Bone conduction thresholds are grossly equal to air conduction thresholds.

*Spondaic words. Recorded version: CID Auditory Test W-2, descending intensity recording.

**Monosyllabic words. Recorded version: Rush Hughes recording, PB-50 Auditory Test. Values are per cent words correctly identified.

***Monosyllabic words. Recorded version: CID Auditory Test W-22. Ira Hirsh recording of modified PB-50 lists. Values are per cent correctly identified.

#Cases previously reported by Walsh and Goodman.[1]

##Cases previously reported by Goldstein, Goodman and King.[10]

of the cerebellopontine angle. (Cases GG, LR, LB and DH of this group were reported originally by Walsh and Goodman.[1]) Surgical confirmation is provided for nine of these; three are presumed on the basis of X-ray and/or related neurologic evidence. There is one case with a confirmed tumor of the third ventricle and there is one case with presumed multiple metastatic brain lesions. The four cases of left hemispherectomy (TW, DS, WM, EW) are the subject of a report by Goldstein, Goodman and King.[10] For these cases the auditory data are reconsidered here as a more convenient way to bring these observations into the general consideration of auditory function in intracranial lesions.

Hearing for pure tones and for speech. If we define a severe loss for pure tones as one in which the hearing loss in the speech-frequency range is greater than about 50 db., we may describe the loss in the group with cochlear disease as varying from moderate to severe. In the group with intracranial lesions hearing loss for pure tones varies from normal hearing to a severe loss. (It should be noted at this point that those lesions in which the hearing loss for pure tones is total are excluded from this study).

For the subjects with cochlear disease, hearing loss for speech was measurable in all cases. The discrmination loss for the Rush Hughes recordings of the PB lists was severe in all cases: per cent words correctly identified ranged from zero to approximately 50 per cent. In all cases the discrimination loss for speech was severe relative to the threshold loss for pure tones. In no case, however, was there a total loss of discrimination for all speech.

In marked contrast to the cochlear-disease group, half of the patients with lesions of the cerebellopontine angle were unable to discriminate any speech delivered to the affected ear. The residual hearing for pure tones in these cases was such that one would expect some discrimination at least for the easier speech signals (spondees or connected discourse). Fig. 1 presents the hearing loss curves for two groups with lesions of the cerebellopontine angle. Group I represents those ears which could discriminate no speech. Some of the losses for pure tones are severe, and we would expect such

curves to relate to severe discrimination losses for Rush Hughes PB lists. We would not, however, expect a total loss of discrimination for all speech. The hearing loss curves for Group II are not remarkably different from the curves in Group I. None of these ears, however, evidenced a total discrimination loss for speech, although with one possible excep-

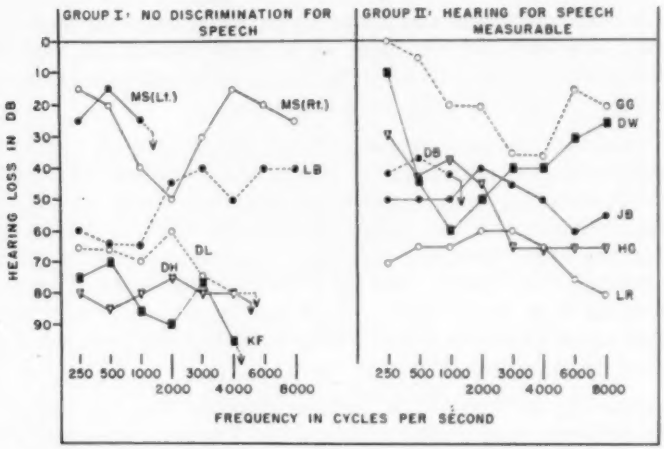

Fig. 1. Air conduction audiograms of two groups of patients with lesions of the cerebellopontine angle. Patients in Group I could discriminate no speech signals delivered to the affected ear. For patients in Group II, although discrimination loss for speech was severe, hearing for speech was measurable. (Bone conduction thresholds are grossly equal to air conduction thresholds.)

tion (HG), they all showed a severe discrimination loss for Rush Hughes PB words. The curve for Case DB is the later curve for one of the cases which earlier showed a discrimination loss for speech associated with normal hearing for pure tones in the speech-frequency range. At this second examination there were no correct responses for PB words, but with pure-tone sensitivity measurable only at 250, 500 and 1000 cycles (hearing loss 35 and 40 db.) the hearing loss for speech was 38 db. Compare this with the curve for Case MS (Lt) in Group I where a similar audiogram relates to a total loss of discrimination for speech. Case DL, shown

in Group I, was re-examined at a later date. At that time, with essentially the same hearing loss for pure tones as on the first visit, he was able to discriminate some spondees (data shown in Table II). For one or more reasons the ears represented in Group I appear incapable of following the rapid frequency, energy and complexity changes necessary for accurate identification of speech signals. Fig. 1 suggests that the explanation for the severity of the discrimination loss for speech for these lesions of the cerebellopontine angle does not appear to lie in the measurements of hearing for pure tones. Nor, as the evidence brought forth in a later section will imply, does the explanation appear related to the presence of loudness recruitment.

Certain of the observations with lesions of the cerebellopontine angle are worthy of further comment. Cases GG and DB show hearing for pure tones within normal limits for the range of frequencies usually considered responsible for discrimination of speech.[16] The discrimination loss for PB monosyllables delivered to the affected ear, however, is severe. It is also interesting in view of the shape of the hearing loss curve and the fact that the opposite ear was normal, that DB complained at that time of difficulty in hearing. The hearing test results at the second examination of this case show the progression of auditory dysfunction. The clinical diagnosis was made at the second date.

For Case MS, Table II presents post-operative data on the right ear. The left VIIIth nerve was sacrificed in removal of the tumor. The right VIIIth nerve, however, was spared at least in part. It is interesting to observe, *1.* the pattern of improvement in pure-tone sensitivity at 2000 cycles and below; *2.* the decrement in sensitivity at 3000 cycles and above, and *3.* the change in ability to discriminate speech. Pre-operatively there was no discrimination for any speech; post-operatively, although there was no discrimination for PB words, hearing loss for spondees was measurable and in agreement with the loss for pure tones.

More a subject for future investigation than for present reporting, is the observation of what appear to be discrimina-

TABLE III.

POSSIBLE CONTRALATERAL EFFECTS IN PATIENTS WITH TUMORS OF THE CEREBELLOPONTINE ANGLE.

Case	Age	Ear	Hearing Loss, Db. Pure Tones, C.p.s., Air Conduction† 250	500	1000	2000	3000	4000	6000	8000	Speech W-2*	Discrimination Score RH**	W-22***	Diagnosis
JS	49	R	(no response to any auditory stimuli)											Right angle tumor confirmed
		L	25	30	30	55	75	80	80	80↓	34	61	—	
BW	52	L	(no response to any auditory stimuli)											Left angle tumor confirmed
		R	0	10	10	10	15	10	25	10	19	59	94	
GR	55	R	(no response to any auditory stimuli)											Right angle tumor confirmed
		L	0	0	5	25	25	15	25	40	20	75	—	
DH	54	R	80	85	80	75	80	80	80↓	80↓	No Discrimination			Right angle tumor confirmed
		L	0	10	15	10	20	20	15	5	20	84	—	
KF	62	L	75	70	85	90	75	95	80↓	80↓	No Discrimination			Left angle lesion presumed
		R	5	5	15	10	10	10	20	25	19	63	—	
DL	52	L	65	65	70	60	75	80	80↓	80↓	No Discrimination			Left angle lesion presumed
		R	–5	0	10	10	35	35	30	20	12	64	—	

†Bone conduction thresholds are grossly equal to air conduction thresholds.

*Spondaic words. Recorded version: CID Auditory Test W-2, descending intensity recording.

**Monosyllabic words. Recorded version: Rush Hughes recording, PB-50 Auditory Test. Values are per cent words correctly identified.

***Monosyllabic words. Recorded version: CID Auditory Test W-22. Ira Hirsh recording of modified PB-50 lists. Values are per cent correctly identified.

tion and/or sensitivity losses for stimuli delivered to the ear contralateral to some tumors of the cerebellopontine angle. Table III presents the pertinent data. (Some of the cases are not considered in the major portion of this study, since the ear on the side of the tumor evidenced a total loss of sensitivity to all stimuli). With the exception of the two known instances of bilateral acoustic tumors (Cases MS and DW), the contralateral ears in the remaining cases were not remarkable. Whether or not the six cases presented in Table III show true contralateral effects is open to question. Edwards and Paterson[3] reported five cases in which contralateral hearing loss was observed and unexplained. Revilla[6] reported five such cases, and Cushing[5] reported three cases.

The observations with the intracranial lesions other than those of the cerebellopontine angle are generally similar, in that with these as with the pontine angle lesions, there is a marked disproportion between the discrimination loss for speech and the hearing loss for pure tones. Here, however, if generalization with diverse lesions and fewer cases is justifiable, the disproportion moves to another part of the scale. That is, the hearing loss for pure tones appears more likely to be zero. This incidentally, as with some of the pontine angle lesions, should serve to emphasize that the integrity of auditory function is not assessed by measurement of thresholds for pure tones alone.

In Case JJ, sensitivity for pure tones was within normal limits bilaterally throughout the entire frequency range. The discrimination loss for speech delivered to either ear, however, was total. There was no evidence of language dysfunction. At surgery a large tumor of the third ventricle was removed.

In the four cases of left hemispherectomy, thresholds for pure tones were normal bilaterally. A discrimination loss for Rush Hughes PB words of the order of 45 per cent was measured for stimuli delivered to the contralateral ear. The entire cortex of the abnormal hemisphere was removed in all four cases. For Auditory test W-22,[11,13] an "easier" version of the PB word test than the Rush Hughes recordings, the speech discrimination scores were approximately normal.

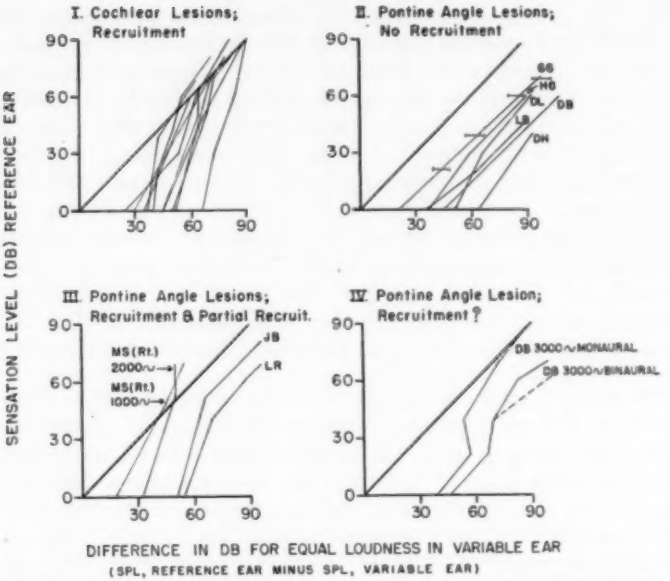

Fig. 2. Loudness recruitment observations. The abcissa values represent the Sound Pressure Level increment required to reach threshold in the variable (pathologic) ear as compared to the reference (better) ear. The difference in decibels required for sensation of equal loudness in the variable ear at levels above threshold is indicated by the distance between the diagonal and a particular equal-loudness curve at a given Sensation Level on the ordinate. The curves shown in IV are for case DB at the initial examination at which recruitment was observed when measured by a monaural loudness balance (3000 c.p.s. versus reference frequency of 2000 c.p.s.) but no recruitment was observed when measured by a binaural equal-frequency (3000 c.p.s.) loudness balance. The broken line represents the result when the tone was set at 118 db. SPL in the pathologic ear as a reference signal and the judgments of equal loudness were taken with the normal ear as the variable.

The auditory findings were essentially the same pre- and post-operatively.

The remaining case (JW) was diagnosed as one of multiple metastatic brain tumors. For tones and for speech delivered to the left ear the responses were normal and reliable. Speech delivered to the right ear, however, was not intelligible except for numbers. The patient reported that the right ear signal (speech) sometimes sounded like speech and sometimes like noise. Hearing for pure tones delivered to the right ear was not measurable because of an extreme

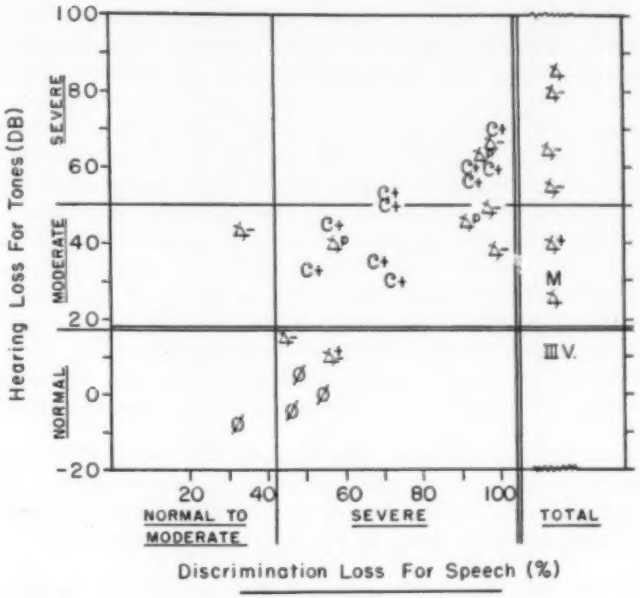

Fig. 3. Discrimination loss for speech plotted as a function of hearing loss for pure tones. Diagnostic category is the parameter. The presence or absence of loudness recruitment is indicated for those instances in which loudness balance tests were made. The results of examinations at two visits are plotted for cases DB, LR and DL (see Table II). The ordinate values represent the average hearing loss for 500, 1000 and 2000 c.p.s. The abcissa values represent the per cent words incorrectly identified with the Rush Hughes PB recordings except for the value shown as greater than 100 per cent. The latter category of discrimination loss for speech represents the result with those patients for whom hearing for speech was not measurable in that they could discriminate no speech (PB's, spondees or connected discourse) delivered to the affected ear (see text).

variability in sensitivity. The tones were heard at moderate hearing loss values, but no threshold measurement could be repeated with a reliability better than about plus or minus 25 db.

Fig. 3 summarizes the relations that appear to exist be-

tween hearing loss for tones and discrimination loss for speech. Examination of the figure indicates that whereas in cochlear disease the discrimination loss for speech is simply severe relative to a given threshold loss for pure tones, in intracranial disease the discrimination loss for speech may be severe but is frequently total for all speech relative to a range of hearing loss for pure tones from normal sensitivity to a severe loss. (Total loss of sensitivity for pure tones is excluded from this study). Further, in intracranial disease, the severity of the discrimination loss for speech does not appear directly proportional to the severity of the hearing loss for pure tones. In lesions of the cerebellopontine angle the discrimination loss for speech is demonstrated for stimuli led to the homolateral ear, in cortical lesions this loss is apparently demonstrated for stimuli led to the contralateral ear.

Loudness Recruitment. Fig. 2 shows the results of loudness balance tests. In the cases defined as non-recruiting, the sound-pressure-level difference at the high sensation levels was approximately equal to the difference at threshold.

In the ten cases with cochlear disease complete loudness recruitment was uniformly demonstrated. In nine of the intracranial lesions where a loudness balance test was indicated and could be carried out, loudness recruitment was absent in six, partial recruitment was observed in two and complete recruitment was observed in one. An interesting, and confusing, observation is the absence of recruitment at the first visit of Case DB when the examination was accomplished with a binaural loudness balance of 3000 c.p.s. vs. 3000 c.p.s., and the demonstration of complete recruitment when 3000 c.p.s. was balanced monaurally against 2000 c.p.s. This phenomenon is unexplained.

Diplacusis. There are nine cases of cochlear disease and nine cases with lesions of the cerebellopontine angle in which adequate pitch balance observations are available. Diplacusis was considered to be present when a marked quality difference (a loss of tonality, for example) was reported or when the difference in frequency required for a judgment of equal pitch was at least 50 cycles.

TABLE IV.

RESULTS OF PITCH BALANCE TESTS.

	DIPLACUSIS		NO DIPLACUSIS	
	Measurable Frequency Difference	Marked Quality Difference	Moderate Quality Difference	No Measurable Frequency Difference
Cochlear Lesions	WZ 500~=350~ JR 1000~=950~ BA 1000~=1030-1050~	CJ Cannot Match	WS* "White Noise Component in Tone"	WS* (Moderate Quality Diff.) VB RE MB JHB
Lesions of the Cerebellopontine Angle	LB 3000~=2300-2500~ LR 2000~=1550-1700~ DL 2000~=1750~	DB "Two Sounds" HG Loss of Tonality GG Cannot Match	MS* "Shrill" JB* "Dull"	MS* (Moderate Quality Diff.) JB* (Moderate Quality Diff.) DH

Table IV presents the data on this measure. In three of the cases with cochlear disease a measurable difference was demonstrated, and in one the quality difference between the two ears was so great as to prevent a pitch match. In the lesions of the cerebellopontine angle a measurable frequency difference was demonstrated in one-third of the total and a quality difference too severe to permit pitch-matching was observed in another third. In other words, diplacusis was observed in a little less than half of the cases with cochlear disorders and in a little more than half of the cases with lesions of the cerebellopontine angle. The other patients with intracranial lesions also gave evidence of pitch dysfunction: Case JW with multiple metastatic brain lesions reported a severe loss of tonality and the four cases of left hemispherectomy reported a difference in quality for pure tones presented to the right ear.

DISCUSSION.

Hearing for Speech. A survey of the pertinent literature reveals less than complete agreement with the present observations. Dix, Hallpike and Hood[17] reported two cases of VIIIth nerve tumor in which "good restoration of intelligibility was obtained with amplification". Eby and Williams[18] reported five cases of lesions of the cerebellopontine angle which showed similar behavior. On the other hand, Liden[19] reported seven such cases which demonstrated remarkably poor discrimination for speech relative to the hearing loss for pure tones. Schuknecht and Woellner[20] reported two cases of VIIIth nerve tumor, one with a moderate threshold loss and the other with essentially normal hearing for pure tones in the speech-frequency range, both of which showed very poor discrimination for speech. Kos[21] reported a case of pinealoma which showed auditory dysfunction similar to that observed in Case JJ. Recently, Dix[22] has referred to observations of Dix and Hood (unpublished). She states ". . . in a significant proportion of . . . cases of deafness associated with tumours of the cerebellopontine angle, restoration of speech intelligibility by means of amplification is not a constant finding". Dix comments further that among the factors

which affect the achievement of good speech intelligibility with amplification is the occurrence in some VIIIth nerve tumors of partial loudness recruitment. On the basis of the present data, however, partial recruitment is not likely to explain the poor speech discrimination observed in intracranial lesions. The cases with cochlear disease presented here, all with complete recruitment, show better discrimination in general than do the intracranial lesions without recruitment, and the two cases with intracranial lesions in this study which demonstrate partial recruitment (LR and JB) are among those with better speech discrimination.

The literature indicates[23,24,25] that in presbycusis, too, the discrimination loss for speech is likely to be severe relative to the hearing loss for tones. Schuknecht[25] states that this auditory dysfunction, referred to as "phonemic regression[23]" in aged patients, results from a loss of neurons in the auditory pathways. Whatever the underlying physiology may be, from a behavioral point of view phonemic regression appears to be in the same family of things as the intracranial lesions reported in this study. The problems in differential diagnosis that are likely to arise from this familial resemblance, however, would appear restricted to those instances in which there may be a question of bilateral intracranial lesions in an older person. Bilateral acoustic tumors, for example, occur primarily in the second decade of life,[3] and unilateral auditory dysfunction at any age would not seem characteristic of presbycusis.

Loudness Recruitment. All of the present cases with cochlear disease showed loudness recruitment. It should be noted, however, that Palva[26] reports, as does Kos,[21] that in some cases of Meniere's disease loudness recruitment, although present in the early course of the disease, is likely to be absent at a later stage when the hearing loss is relatively stable. This appears consistent with Schuknecht's[25] statement that recruitment requires an incomplete sensory lesion in which there is a partial loss of hair cells and a relatively normal number of ganglion cells remaining within the fields of excitation. Since the present study required cases in which marked remission of hearing loss was measured, the con-

sistent observation of recruitment in this series cannot be taken as contradictory of Palva's and Kos' observations.

The presence of loudness recruitment in intracranial lesions requires comment. Dix and Hood[27] reported recruitment in 8 per cent of a series of 72 lesions of the cerebellopontine angle (57 confirmed) and explained that its presence is "connected with hair cell changes resulting from occlusion of the cochlear vascular supply". The series being reported now is perhaps too small for proper comparison with that of Dix and Hood on the question of the incidence of recruitment in lesions of the cerebellopontine angle. The incidence would be 11 per cent with complete recruitment, and if the two cases with partial recruitment are added it would be 33 per cent. Of greater interest is the lack of consistent observation at surgery, of interference with the cochlear blood supply. Such observation was made in one of those cases (LR) with partial recruitment. This observation was not made in the other case with partial recruitment (JB), nor was it made in the case with complete recruitment (MS, Rt.). On the other hand, the pathology in one of the pontine angle lesions (HG) in which no recruitment was demonstrated was diagnosed as a vascular anomaly of the internal auditory artery. At this stage of development it seems reasonable to take the position that in lesions of the cerebellopontine angle recruitment may be present but is characteristically absent, that when recruitment is present it may be explained by interference with the cochlear vascular supply or its presence may be unexplained.

Diplacusis. Diplacusis, as defined and measured in this study, does not distinguish between intracranial lesions and cochlear lesions. Thus the present observations do not agree with general clinical opinions which tend to associate diplacusis with cochlear lesions. The prevalent conception appears to arise from the reports of Shambaugh, Jr.,[28] Davis, et al.,[29] Eby and Williams,[18] and Williams.[30] On the other hand, Schuknecht and Woellner[20] reported that one of their cases with a tumor of the cerebellopontine angle experienced a loss on tonality on the affected side. In addition, Azzi[31] has investigated the frequency difference limen with clinical mater-

ial. He used a frequency modulation technique, and he observed that the difference limen was not increased in cases with cochlear disease but was increased in cases with lesions of the VIIIth nerve.

Diagnostic Implications. Most of the experience with subtentorial lesions reported is with lesions of the cerebellopontine angle. Without additional experience with other brain-stem loci no inferences can be drawn about the similarities or differences in auditory function that may obtain between lesions of the cerebellopontine angle and other lesions of the brain-stem. It would appear necessary, therefore, in applying the present observations on lesions of the cerebellopontine angle to the prediction of anatomic site, to speak of the probable site of disturbance in a given case as "brain-stem" in general rather than "cerebellopontine angle" in particular. Similarly, by reason of the limited experience with lesions other than tumors, it seems desirable to avoid predicting the pathology of a suspected lesion.

In discussing the auditory signs which may at this time be designated as indicative of intracranial lesions it is pertinent to comment that the author is not suggesting to the neurologist or neurosurgeon that the diagnosis of intracranial disease be made on the basis of audition alone, although there are cases of VIIIth nerve tumor for which this accomplishment is reported.[27,32] Rather, the observations reported suggest the nature and direction of cooperation between the neurosurgeon and the audiologist. It is, however, interesting to speculate on the accuracy of clinical diagnosis if the only information available to us were measures of auditory function. In this frame of reference, study of Fig. 3 suggests the following:

1. Those patients with an inability to discriminate any speech (total discrimination loss for speech in the context of this report) associated with either a moderate or a severe hearing loss for tones would be diagnosed as having a lesion of the brain-stem. The presence of loudness recruitment would not negate this diagnosis; the absence of loudness recruitment would confirm it.

2. Those patients with a severe discrimination loss for speech relative to thresholds for pure tones within normal limits would be diagnosed as having an intracranial lesion. The site of the lesion could be either the cerebral cortex contralateral to the ear which yielded these anomalous responses or the ipsilateral brain-stem. The general diagnosis of intracranial disease would be confirmed by the absence of loudness recruitment (if a loss for tones out of the speech range permitted exploration for recruitment); the presence of loudness recruitment would not deny the diagnosis. The possibility for differentiation of these loci would appear to lie in the relations between other auditory functions: the discrimination loss for speech in the cortical lesions is demonstrated for "difficult" speech (Rush Hughes PB's, Bocca's distorted speech[9]), but not for "easy" speech (spondees, W-22 PB's). In the lesions of the brain-stem, there appears to be some discrimination loss for "easy" speech (thresholds for spondees appear greater than would be predicted from the loss for pure tones) and the discrimination loss for the PB monosyllables seems to remain severe even with the easier (W-22) version of this test. The degree of confidence for this differentiation is not yet high.

3. Those patients with a severe discrimination loss for speech and a moderate to severe hearing loss for tones may have either a lesion of the brain-stem or the end-organ. If loudness recruitment is absent we would assume, with some probability of being in error, that the disturbance is in the brain-stem. On the other hand, the presence of either partial or complete loudness recruitment would not rule out the possibility of brain-stem disease.

It appears then, that on the basis of the relations between several measures of auditory function, a clinical diagnosis of intracranial disease can be made with reasonable certainty in some situations. For those situations where the auditory findings are ambiguous and where the site of the intracranial lesion is not differentiated with precision, further inquiry is indicated with refinements of these measures and with measures of auditory function other than those discussed in this study.

SUMMARY AND CONCLUSIONS.

Audiologic information on 18 cases on which neurosurgical or neurologic evidence is available has been presented. Some relations between auditory function and intracranial lesions have been described and compared with lesions of the cochlea. On the basis of the evidence reported in this study, the following generalizations may be made about auditory function in intracranial lesions:

1. While in cochlear disorders there is a discrimination loss for speech which is characteristically severe relative to a measured threshold loss for tones, in intracranial lesions, on the other hand, there is a severe discrimination loss for speech which appears independent of the threshold loss for pure tones: the threshold loss will vary from normal sensitivity to a severe loss (total loss of sensitivity for pure tones has been excluded from this study) while the discrimination loss for speech will vary from a severe loss to a total loss. In addition, in intracranial lesions, the severity of the discrimination loss for speech is not necessarily directly proportional to the severity of the threshold loss for tones. In unilateral lesions of the brain-stem the discrimination loss for speech appears for stimuli presented to the ipsilateral ear, in unilateral cortical lesions this loss appears for stimuli led to the contralateral ear.

2. Loudness recruitment is consistently present in those cochlear lesions which are characterized by remissions in hearing loss; loudness recruitment is not characteristically absent in intracranial lesions.

3. Diplacusis, defined as a loss of tonality, or as a difference in pitch between the two ears in response to stimuli of equal frequency, is as likely to occur in intracranial disease as in cochlear disease.

Within the limits of the examination procedures reported in this study, the following auditory signs may at this time be taken as indicative of an intracranial lesion:

1. An inability to discriminate any speech associated with a moderate to severe hearing loss for tones suggests a lesion

of the brain-stem. The presence of loudness recruitment does not deny the diagnosis; the absence of loudness recruitment confirms it.

2. A severe discrimination loss for speech associated with thresholds for pure tones within normal limits suggests an intracranial lesion. The present information does not permit prediction of the site of the lesion: it may be the contralateral cerebral cortex or the ipsilateral brain-stem. If discrimination loss for speech remains severe with relatively "easy" speech signals, there is a probability that the lesion is in the brain-stem. If examination for loudness recruitment is possible, the absence of recruitment confirms the diagnosis; its presence adds no information.

3. A severe discrimination loss for speech associated with a moderate to severe hearing loss for pure tones is an ambiguous sign: it suggests a lesion of the brain-stem or of the cochlea with equal probability. In such a case, the presence or absence of loudness recruitment is similarly ambiguous.

The suggestion is made that further inquiry into the relations between several measures of auditory function with more refined measures and with measures of function other than those reported may resolve the ambiguities and differentiate the site of intracranial lesion with greater precision.

ACKNOWLEDGMENTS.

The author expresses his gratitude to Dr. Henry G. Schwartz, Professor of Neurosurgery, Washington University School of Medicine, for his encouragement and cooperation in this study. Thanks are due also to Drs. Robert B. King and Frederick W. L. Kerr of the Division of Neurosurgery, Washington University School of Medicine, and to Drs. Ira J. Hirsh and Robert Goldstein of Central Institute for the Deaf for their many helpful consultations in the preparation of this report.

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AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

The American Association for Cleft Palate Rehabilitation will hold its 16th Annual Convention at the St. Francis Hotel, San Francisco, Thursday, Friday and Saturday, April 24, 25 and 26, 1958.

This Association is composed of medical, dental and paramedical specialists who are interested in the rehabilitation of persons with cleft lips and palates. For further information write: Dept. of Otolaryngology, University of Iowa Hospitals, Iowa City, Iowa.

OCCUPATIONAL HEARING LOSS.*

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Occupational hearing loss can be defined as a hearing impairment in one or both ears, partial or complete, arising in, during the course of, or as the result of one's employment. It may occur suddenly as the result of traumatic injury, intense blasts or explosions, or gradually due to prolonged exposure to excessive noise levels.

Direct trauma to the ear usually results from blows, foreign objects and burns due to sparks and molten metal. Local trauma may be followed by perforation of the ear drum and infection. Conduction impairments resulting from trauma are usually unilateral and respond to proper treatment. Direct trauma to the head, such as concussions or fractures, can result in a nerve type hearing loss, the hearing tests revealing audiograms very similar to those found in cases due to noise exposure.

The term, "acoustic trauma," is often used to include noise-induced hearing loss as well as the sudden hearing impairment resulting from intense blasts, explosions and gunfire. These conditions, however, should be described by separate terms in order that one may logically distinguish between them. Acoustic trauma should be reserved for the immediate hearing injury produced by one or a few exposures to very intense sounds such as blasts or explosions.

Industrial noise-induced hearing loss is used to describe the accumulative loss of hearing always of the nerve type that develops over a period of months or years of employment in hazardous noise levels.

There has been a growing interest in the problems of industrial noise and occupational hearing loss in recent years.

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*Presented at the Sixth International Congress of Otolaryngology, Washington, D. C., May 9, 1957.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication June 1, 1957.

The subject has been of particular interest and concern to the otolaryngologist. Several papers and a panel discussion on this topic were presented at the Pan American Congress in Mexico City in 1954.[1]

The effects of industrial noise have been one of the aspects of this problem which have come under scrutiny over the past few years. Generally speaking, they can be divided into the auditory and non-auditory effects. The auditory effects of intense industrial noise are: it causes temporary and permanent hearing loss; also it interferes with communication. The non-auditory effects of industrial noise are vague and ill-defined and at present not scientifically established.

The otolaryngologist, by virtue of his special training and wide experience in hearing problems, is frequently called upon for advice in questions pertaining to occupational hearing loss. Generally speaking, his opinions and guidance are needed in planning hearing conservation programs. These programs are well covered in the recent "Guide for Conservation of Hearing in Noise",[2] published by the Subcommittee on Noise in Industry of the Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology. Much study and effort have gone into this publication, and I recommend its use by all who are confronted with the noise problem. Copies of the above mentioned guide may be obtained from the Director of Research of the Subcommittee on Noise in Industry.

I mention some personal medical-legal experiences and observations in regard to the evaluation of claims for alleged loss of hearing due to noise-exposure. The first question to be answered after a claimant has been examined is, "Does a hearing loss exist?"—*i.e.*, the otolaryngologist is asked to determine whether or not the claimant actually has lost hearing ability that he formerly possessed. This is done by proper history and otological examinations, and a battery of hearing tests such as pure-tone air and bone conduction audiometric tests, speech tests, tuning fork tests and careful observation of the claimant's speech and hearing during the course of the examination.

Before forming an opinion that the claimant has sustained a hearing loss due to noise exposure the otolaryngologist must be satisfied that: *1.* the claimant has the usual nerve type hearing loss that accompanies noise-exposure; *2.* the sound survey studies of the working areas reveal the noise is of a type and of sufficient intensity to cause hearing loss; *3.* there has been sufficient exposure time and, *4.* other local and medical causes for hearing loss have been ruled out by proper history, otological examinations and hearing tests.

Too often I have heard physicians describe the results of a claimant's history and hearing tests, both of which were insufficient to permit the differential diagnosis of noise-induced hearing loss. I have also heard testimony before industrial commissions on workers employed in noisy industry for many years in whom the hearing loss was attributed to high blood pressure, allergy, infection, heart disease, diabetes, or advancing age simply because the hearing tests disclosed a high-frequency hearing loss. The otolaryngologist is not justified in ascribing the hearing loss to such causes completely ignoring the fact that the worker has been continuously employed at a punch press, riveting machine or drop hammer for the past ten or 20 years; nor is he justified in attributing the hearing loss to occupational origin when the history (includes military and occupational), otological examination, and hearing tests reveal findings consistent with otosclerosis, middle ear disease and conductive hearing loss.

The otolaryngologist who appears as a medical witness in these cases must be an impartial, disinterested expert who will assist the courts and commissions in resolving the medical problem at hand. He should testify honestly, authoritatively, thoroughly, and with conviction. The role of the medical man is to clarify not to confuse, and above all, he should not attempt to cloud the issues. His statements should be based upon reasonable medical opinions and conclusions and not upon mere speculation. He must be objective and scientific and should not concern himself with the social and economic aspects of the problem. Decisions as to when, how much, or under what condition compensation is to be

paid for loss of hearing are not matters for the medical man to decide. These are the function of the commissions and courts.

Some of the more important medical-legal questions which have arisen are: *1.* At what level of noise intensity and over what period of exposure does damage to hearing take place? *2.* What was the worker's pre-employment hearing status? *3.* What steps should be used to determine hearing loss? *4.* What formula should be used to compute hearing handicap? *5.* How can we decide when a hearing loss is permanent? *6.* What consideration should be given to non-occupational hearing loss that accompanies age? The Sub-committee on Noise in Industry is active in trying to find answers to these thorny questions.

Numerous legal questions as to the intent and interpretation of existing compensation laws concerning noise-induced hearing loss are gradually being answered by authoritative decisions of the higher courts of various States. This has already occurred in the States of Wisconsin, New York, Missouri and in the Federal Courts of New York.

One of the most important controversial medical-legal developments is that pertaining to the methods for determining hearing loss and for converting this hearing loss into terms of hearing handicap. This brings up the questions, "What is hearing loss and when does a hearing loss become a hearing handicap?" And also, "What kind of tests do you use and how do you determine from these tests the amount of handicap?" A discussion of these questions is found in the report of the Committee of Eight, entitled, "Principles for Evaluating Hearing Loss," published in the *Journal of the American Medical Association*, 157, April 16, 1955.[3]

The State of Wisconsin, which has been a leader in Workmen's Compensation, passed specific legislation in 1955 dealing with hearing loss resulting from industrial noise exposure.[4] The medical aspects of the problem were referred to a Medical Advisory Committee appointed by the Wisconsin Industrial Commission. Of particular interest was the medical

TABLE I.

HEARING DISABILITY TABLE.

Average Decibel Loss	% of Compensable Hearing Loss	Average Decibel Loss	% of Compensable Hearing Loss
17	.8	49	53.3
18	2.2	50	55
19	3.6	51	56.7
20	5	52	58.3
21	6.7	53	60
22	8.3	54	61.7
23	10	55	63.3
24	11.7	56	65
25	13.3	57	66.7
26	15	58	68.3
27	16.7	59	70
28	18.3	60	71.7
29	20	61	73.3
30	21.7	62	75
31	23.3	63	76.4
32	25	64	77.8
33	26.7	65	79.2
34	28.3	66	80.6
35	30	67	82
36	31.7	68	83.4
37	33.3	69	84.8
38	35	70	86.2
39	36.7	71	87.6
40	38.3	72	89
41	40	73	90.4
42	41.7	74	91.8
43	43.3	75	93.2
44	45	76	94.6
45	46.7	77	96
46	48.3	78	97.4
47	50	79	98.8
48	51.7	80 and over	100

Method—Add the pure tone air conduction losses in decibels for the three (3) frequencies 500, 1000 and 2000 c.p.s. and divide by three (3) to obtain average decibel loss. Find this average in the vertical column on the left (marked average decibel loss) and the percentage of compensable hearing loss for that ear is given directly opposite.

For Binaural Percentage of Hearing Loss use the following formula:
Four times the percentage of lesser loss;
Add the Percentage of greater loss;
Divide total by five.

The resulting percentage is the loss of hearing in both ears and is applied to the schedule allowance for loss of hearing in both ears.

group's recommendation of a method for determining hearing loss and hearing handicap. The Wisconsin method is shown above (see Table I).

SUMMARY.

Occupational hearing loss is assuming increasing importance in our everyday life. It is the outgrowth of our rapidly expanding industrial development. The industrial noise problem is a complicated one, because it involves many technical and professional fields. In my opinion the otolaryngologist should become interested in and study the many facets of this problem. He should consult with the industrial hygienist, the plant physician and other necessary personnel, and if in the medical-legal evaluations he is objective, impartial, scientific and "sticks" to the medical facts, he can play a vital role in the problem of occupational hearing loss.

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DECANNULATING THE TRACHEOTOMIZED PATIENT WITH POLIOMYELITIS.*†

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In recent years the use of a tracheotomy in bulbar poliomyelitis and certain other debilitating diseases has been widespread, although the indications differ with various writers. Briefly the usefulness of a tracheotomy in bulbar poliomyelitis may be listed as follows:

1. A by-pass around uncontrolled nasopharyngeal secretions.

2. A readily accessible portal to the lower respiratory tract.

3. The reduction of the dead air space in the respiratory tract.[1,2]

4. The reduction of the resistance to the in- and out-flow of air through the upper respiratory tract.[1]

5. Provision of any airway in the event of either glottic spasm or bilateral paralysis of the vocal cords.

The purpose of this paper is to discuss some factors which must be considered before decannulation of cases of poliomyelitis. The known facts concerning the physiology of a normal cough will first be presented, and then several cases, illustrating the unfortunate sequellae when decannulation was done too early, will be summarized. Finally there will be a discussion of ways to assess the quality of the cough and thus avoid the pitfall of too early decannulation.

In general there are only two principal physiologic considerations when decannulation of a case of bulbar poliomyelitis is contemplated: first that the patient be able to

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*Presented in part as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1957.

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Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 18, 1957.

swallow effectively and efficiently; second, that he have a "good enough" cough to enable him to keep his lower respiratory tract clean. There is also a psychic consideration in that the patient may become "dependent" upon the artificial airway. He is unnecessarily fearful of removal of the tracheotomy tube, consequently once it is deemed physiologically safe to decannulate, it should be done at once.

The nasopharyngeal secretions may amount to as much as 1,000 to 1,500 cc. per day (Proetz). It is apparent that if the patient cannot handle these effectively by expectoration and swallowing there is grave danger that they may be aspirated. It is also desirable that he be able to swallow 2,000 cc. or more of fluids per day by mouth so as to maintain his fluid and nutritive requirements. It would seem probable that if he can effectively swallow his nasopharyngeal secretions he also can learn to handle the daily requirements of food and drink. Whether an individual swallows effectively and efficiently is much easier to determine than is the second important consideration prior to decannulation; namely, the effectiveness of his cough.

The purpose of a cough is to expel foreign or exudative material from the lower respiratory tract. It is aided in this by ciliated cells which line the trachea and bronchi down to the alveoli. A "good" cough will carry such foreign or exudative material upward to the hypo-pharynx where it is either swallowed or expectorated. In the event that the cough is ineffective the ciliary action alone, as indicated by the following case histories, is unable to keep the lower respiratory tract clean. If, however, a tracteotomy has been performed, the lower respiratory tract may be reached readily and the exudate repeatedly aspirated until the lower respiratory tract is grossly clear. The difficult decision is to determine when the cough is sufficiently strong so that it, along with the cilia, can keep the lower respiratory tract clean. When this occurs then it is safe to close the tracheotomy.

NORMAL COUGH.

A review of the dynamics of a normal cough, about which the literature contains scant information, will be helpful in

determining the optimum time of decannulation in bulbar poliomyelitis. In general a cough requires that the intrapleural pressure be elevated and then suddenly released so that there is a vigorous expiratory blast of air which will carry with it the exudate or other material from the lower respiratory tract. To do this the glottis must be capable of being tightly closed and then suddenly released.

Various factors are involved in creating the increased intrapleural pressure. First the lungs must be filled with air by the combined action of the intercostal muscles, the elevators of the ribs, the accessory inspiratory muscles and the diaphragm. During this *inspiratory phase* the chest dilates and elongates, thus producing a strongly negative intrapleural pressure. When the chest dilates, the lung expands and air rushes into it, neutralizing a part of the negative intrapleural pressure.

There now is an enlarged, rigid thorax and a lung filled with a large amount of air. To bring this under pressure it is necessary to close the upper and lower ends. Contraction of the glottis accomplishes the former, but the latter is less well understood. It will be more fully discussed subsequently when the action of the diaphragm is considered.

Under the action of the expiratory muscles, the ribs and sternum are drawn downward, causing a marked reduction in the capacity of the thorax and an increase in pressure on the air which is trapped in the lung by closure of the glottis. This is termed the *compressive phase.*

THE DIAPHRAGM.

During the inspiratory phase of the cough the participation of the diaphragm is obvious. By its contraction it elongates the thorax and increases the space available for air to be drawn into the thorax. When paralyzed there is a reduction of air drawn into the chest during the inspiratory phase.

Coryllos[3] was the first to designate the importance of the diaphragm during the second or compressive phase of the

cough. When the abdominal muscles contract pressure is brought to bear on the abdominal viscera. If we assume the urethra, anus, and uterine cervix are tightly closed this force is transmitted upward toward the thorax. The contraction of the diaphragm creates a force downward opposite to that of the abdominal muscles; thus we have two antagonistic forces, and their combined action creates a rigid floor to the thorax.

Paralysis of the anterior abdominal muscles or the diaphragm reduces the rigidity of the floor of the thorax during a cough and reduces its efficiency. The effect of the loss of the anterior abdominal muscles is much greater. In this case increasing the intrapleural pressure merely causes the abdomen to balloon outward. A cough, in these circumstances, is ineffective and would be akin to trying to jump upward in an elevator which was going down. The pitifully poor cough we see in patients recovering from poliomyelitis is almost always due to the lack of useful anterior abdominal musculature.

Paralysis of the diaphragm alone does not prevent efficient coughs. The chief result is a decrease in the amount of air drawn into the chest during the inspiratory phase, and thus a lessened amount available to expel during the expulsive phase of a cough. To this extent the efficiency of the cough is decreased.

THE EXPULSIVE PHASE.

Once the air trapped in the lung is under increased pressure the glottis is suddenly opened. This permits a vigorous expulsive blast of air which will carry with it the exudate or other materials from the lower respiratory tract.

Matheson, et al.,[4] have likened the lung to a simple mechanical bellows. There are three factors which operate on such bellows, namely, the size, the forces operating it and the resistance. In the case of the lung, the size is measured by the vital capacity and the forces operating on the lung by the maximum expiratory pressure. The resistance which the upper and lower respiratory tract offers is difficult to meas-

ure. Similarly, in the case of a cough, the patient must be capable of inhaling a large amount of air as well as bringing it under increased pressure by depressing the thoracic cage against a rigid floor to the thorax before suddenly releasing it through the glottis.

Over the years the dynamics of a normal cough have not received the attention they deserve. A great deal of attention has been directed toward the vital capacity both of adults[4,5,6] and adolescents.[7,8] The vital capacity measures only a static volume, namely, the volume of air available for expulsion during a cough; it does not measure the muscle force necessary to expel the air.

In 1951 Gaensler[9] described the usefulness of the one second vital capacity. By this is meant the amount of air the subject can forcefully exhale in a measured time (usually one second) after a maximum inspiration. He found that normal volunteers were able to expire 83 per cent of the total vital capacity in the first second, 94 per cent in the first two and 97 per cent in the first three seconds. These capacities correlated very well with the more cumbersome maximum breathing capacity, the air velocity index and the ratio of the reserve volume to the total capacity. The use to which the one second capacity may be put in determining muscle power available for coughing will be discussed later.

Matheson, et al.,[4] measured the maximum expiratory pressure by having the subject, after a maximum inspiration, exhale as forcefully as he could into the rubber tube of an aneroid sphygmomanometer. In 54 medical students the mean of the fourth and fifth trials was found to be 140.80 mm. Hg. (standard deviation was 32.1).

Whittenberger and Mead[10] recorded the intrapleural pressures during a vigorous cough in three normal human subjects by means of balloons placed in the esophagus. They found that the intraesophageal pressure builds up rapidly by the expiratory effort while the glottis is closed. At approximately 80-100 mm. Hg. the glottis opens and the pressure continues to climb at a lower rate for a short while. In normal subjects the peak pressure then rapidly declines

to essentially atmospheric levels. The whole cycle constitutes approximately 0.4 seconds. Coryllos,[11] with needles inserted into the intrapleural space, found pressure of 80 to 100 mg. Hg. similarly before the glottis opened.

Whittenberger and Mead[10] also found by measurements at the mouth with pneumotacograph, that the average expired volume (three normal subjects) during a cough was approximately 1.94 liters. The estimated linear velocity for the three cases was 96 meters per second. Barach[12] found that the maximum expiratory flow rate in one normal subject during a natural cough was 4,090 cc./second or 245.4 liters per minute.

Rohrer[13] in 1921 calculated the velocity of air during a cough. In the respiratory bronchioles it varied from 0.5 to 2 meters/sec., and at the glottis from 50 to 120 meters/sec. The latter equals 360 feet/sec. If the velocity at the glottis was 300 feet/sec. this would be equivalent of 20 miles/hour.

Jannison[14] using high speed photography, measured the speed of droplets emitted from the mouth during a vigorous *sneeze* and recorded velocities as high as 152 feet per second (46 meters/sec.). The droplets measured were two inches from the mouth. Their speeds would have been greater if the measurements had been taken closer to the source. Hobby[15] has recently stated that during a cough particles are propelled through the air at a rate of 150 to 160 feet per second.

In health it does not seem that resistance is of much importance in the dynamics of cough. Carter and Giuseffi[1] measured the resistance offered by the upper respiratory tract in dogs. They found the intrapleural excursion during quiet breathing to vary from approximately -4.6 to +0.6 or an excursion of 5.2 cm. of water. After side-tracking the upper respiratory tract by a tracheotomy the excursion was reduced to approximately 2.6 cm. of water. Similar results were found in studies on two humans. It seems that in a limited number of observations a tracheotomy had reduced the resistance to the in- and out-flow of air. The pressures involved in a normal cough are much greater than this.

If, however, the muscle power for breathing is much reduced so that the vital capacity is close to the expected normal tidal volume for the patient in question the resistance may be of great importance. Such a patient after decannulation may not possess the additional muscle force necessary to overcome the additional resistance either for coughing or for breathing.

CASE REPORTS.

These case reports are selected from individuals cared for at the Evanston Hospital, Evanston, Ill., during the years 1950-54, inclusive. During this period there were 135 cases in which a diagnosis of polioencephalitis, bulbospinal poliomyelitis, or bulbar poliomyelitis was made. Of these 70 were subjected to tracheotomy. In the following case reports the formulae used to predict the expected vital capacities of healthy adults are those described by Baldwin et al.[6] They are as follows:

Males—[27.63 - (0.112 x age in years)] x height in cm.

Females—[21.78 - (0.101 x age in years)] x height in cm.

The actual vital capacity of the patient in question was determined in all cases by the Bennett ventilation meter.

R.K., male, age 36, height 5'6": This patient was admitted in Sept., 1952, and a diagnosis of bulbar poliomyelitis made. On the third hospital day a tracheotomy was done and closed ten days later. The recovery was uneventful until Oct., 1953, when an upper respiratory infection occurred. It soon became apparent that his cough was insufficient to keep his lower respiratory tract clean, so the tracheotomy was reopened. The recoverey was uneventful.

Comment: The highest vital capacity obtained in this patient was 725 cc., which is 18.6 per cent of the capacity predicted for a person of this height by the formula of Baldwin. It appears that the tracheotomy was closed before a sufficiently effective cough had developed.

R.W., male, age 32, height 5'9": In Sept., 1952, the patient was admitted to the hospital, and a diagnosis of bulbar poliomyelitis was made. Tracheotomy was performed on the second hospital day and removed 32 days later. An uneventful recovery ensued. It was noted at this time that he had "virtually no cough" and that his greatest vital capacity was 350 cc., which represents 8.4 per cent of the expected capacity as predicted by the Baldwin formula.

In January of 1953, he was again admitted with a respiratory tract infection. Immediate bronchoscopic removal of the secretions in the lower respiratory tract was followed by marked improvement, so further bronchoscopic therapy was omitted. Re-opening of the tracheotomy was considered, but the idea abandoned because of the marked improvement. On the third hospital day the patient suddenly died. Post-mortem examination showed a large "rope" of mucus, approximately 1.5x1x10 cm. straddling the carina, thus occluding both bronchi.

Comment: More attention should have been paid to the poor, ineffective cough, as evidenced by the low vital capacity displayed by this patient.

J.C., male, age 31, height 5'10½": J.C., was admitted in August, 1952, and a diagnosis of bulbar poliomyelitis made. A tracheotomy was performed on the fourth hospital day, closed on the twenty-sixth and was followed by an uneventful recovery. His vital capacity was 250 cc. or 5.9 per cent of normal.

In November, 1952, however ,the patient developed a respiratory tract infection with a right-sided pneumonia and atelectasis developing. Repeated bronchoscopy failed to keep the lower respiratory tract clear, and the tracheotomy was reopened with a rapid and uneventful recovery following.

Comment: In retrospect it would appear that a vital capacity of 5.9 per cent of normal should have been sufficient evidence of an ineffective cough to preclude closure of the tracheotomy.

W.J., male, age 31, height 5'11": The patient was admitted in Sept., 1953, and a diagnosis of bulbar poliomyelitis made. He was discharged in May, 1954, with the tracheotomy tube in place but corked most of the time. His greatest vital capacity was measured at 450 cc. or 10.5 per cent of normal.

In June, 1954, he was readmitted with a respiratory tract infection but with an uneventful recovery. He was discharged again with the tracheotomy *in situ* but corked most of the time.

Comment: This patient has been safely handled by leaving a small tracheotomy tube *in situ,* available to mechanical suction or inspection of the lower respiratory tract. With the cannula corked he has a normal voice. If the vital capacity reaches 30 to 40 per cent of normal decannulation will be considered.

V.W., male, age 47, height 6'0": The patient was admitted in Sept., 1953, a diagnosis of bulbar poliomyelitis made, and a tracheotomy performed on the day of admission. His recovery was uneventful except for:

1. Ankylosis of the temporomandibular joints permitted separation of the central incisor teeth of one cm. only, and

2. The almost complete failure of return of the ability to swallow. Consequently he still receives food by a stomach tube. His vital capacity has returned to approximately 30 per cent of normal. The tracheotomy

tube is still *in situ*, corked much of the time. Drooling of the nasopharyngeal secretions is excessive.

DISCUSSION.

The objective measure of what constitutes a "good" and effective cough is not easily determined. Of the various continuous events which together constitute a cough, the most readily measured is the vital capacity and the determination of that volume is the criterion used in the cases reported here. It is clear that the quantity of air inhaled is the same that is available for forceful exhalation during a cough. If the individual is capable of a vital capacity of only a few hundred cc. his cough will be poor. On the other hand a large vital capacity will provide a large quantity of air for expiration, and the efficiency of the cough will be improved. If, in addition to a large vital capacity, there is adequate muscle force available to bring this inhaled air under pressure before being suddenly released through the glottis, it will be still more efficient. From my observations it appeared that vital capacities below 30-40 per cent of predicted normal for that person implied an inefficient cough and above that figure a cough which, together with the cilia, probably could keep the lower respiratory clean.

It seems, however, that vital capacity measurements would be more useful if a concomitant measurement of the muscle force available for coughing was made. The use of the maximum expiratory pressure, as suggested by Matheson et al.,[4] to gauge the muscle force was tried but was found unreliable. To be accurate the subject must keep the glottis open as he blows into the aneroid sphygmomanometer. Failure to do this, particularly in children, is difficult to detect. The ultimate usefulness of this, along with vital capacity studies to evaluate the cough, is yet to be determined.

It seemed probable that the timed vital capacity as suggested by Gaensler[16] and others might be useful in evaluating the muscle force. The technique of its use has been discussed. In healthy volunteers 83 per cent of the maximum inspiration was forcefully expired in the first second. The usefulness of the one second vital capacity seems more credit-

able, because it correlates well with the maximum breathing capacity and the air velocity index. How it correlates with the maximum expiratory pressure is still unknown. Measurement of the one, two, or three second vital capacity is readily taken by the Vitalometer.[17] This instrument is easily portable and requires but a few seconds to perform the test.

Variations in the normal-timed vital capacities are caused by what Gaensler terms obstructive and restrictive defects. In the former there is an obstruction to the free flow of air in the bronchial lumina; asthma is an example. The one second capacity is decreased in obstructive defects whereas the vital capacity remains relatively normal.

In restrictive ventilatory defects there is a loss of aerated lung parenchyma such as may occur in lung tumors, atelectasis, etc. The vital capacity faithfully records these defects whereas the one second capacity may be normal. Gaensler includes the weakened musculature of poliomyelitis, myesthenia gravis, and phrenic nerve paralysis in this group.

In a case recovering from poliomyelitis, however, we have a patient who usually has no obstructive defects such as asthma and has no restrictive defect other than lack of muscle force for respiration and coughing. It seemed worth consideration that the one second capacity of such an individual would vary directly with the muscle force available for expelling the air. Examination of patients other than those whose case histories given here and whose cough had been weakened by poliomyelitis revealed a one second capacity of 80 per cent, which is essentially normal. The "Vitalomotor," however, was found to be a much more reliable indicator of the total vital capacity than the Bennett meter used in the cases reported. The inertia and friction of the Bennett meter reduces its acuracy at low volumes.

Of course, the intrapleural pressure may be measured during a cough by inserting a needle through the chest wall into the pleural space, or by the transmitted pressure on a balloon placed in the esophagus. These procedures are too complicated for repeated use on convalescent patients.

The pneumotachogram gives a great deal of information about the velocity of the air expelled during the cough, but again is too difficult a procedure for clinical application.

Several things can be done clinically to increase the force of a cough. The use of an abdominal binder of the scultetus type increases the rigidity of the floor of the thorax, or manual pressure on the abdomen during an attempted cough accomplishes the same thing. By providing a more rigid floor to the thorax the cough is improved.

An individual whose vital capacity is low may "inhale" 600 cc. or so by the method of "frog breathing" described by Dail.[18] A poor cough may be somewhat improved in this way.

The use of mechanical coughing devices has been described by Barch[19] and his co-workers. They have not been particularly useful in the author's hands.

CONCLUSION.

It is apparent from the case histories presented that removal of the tracheotomy tube in a case of bulbar poliomyelitis before an effective cough has developed may be followed by unfortunate sequelae. On the contrary, the patient should not be decannulated until his cough (along with the ciliary activity) is capable of keeping his lower respiratory tract clean.

The ability to swallow is the first consideration when contemplating decannulation. Once this has returned to the point where the nasopharyngeal secretions as well as the daily requirements of fluid and solid food can be swallowed without spillage either into the nose or trachea, decannulation may be considered.

A "good" cough is the second consideration and is dependent upon several factors. First is the vital capacity which is a static volume. From the information in this report it would appear that the vital capacity should be 30 to 40 per cent of normal before decannulation. For example, in an adult male whose theoretical vital capacity should be 4,500

cc., decannulation can be considered after the vital capacity reaches approximately 1,300 to 1,500 cc. Another important factor in the production of an effective cough is the ability to increase the intrapleural pressure, and this is dependent upon the muscle force available. No adequate measure of this dynamic aspect of a cough has been found. The use of the maximum expiratory pressure measurements may be useful in doing this.

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ANALYSIS OF BLOOD AND VASCULAR FACTORS IN THE PROPHYLAXIS OF TONSILLO-ADENOIDAL HEMORRHAGE.*†

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In an effort to reduce the incidence of hemorrhage following surgery upon the tonsils and adenoids, a study of the problem is undertaken with careful scrutiny of all the factors involved. It is recognized that hemorrhagic states exist which cause bleeding upon surgical trauma. The older theories of blood coagulation have been discarded as inadequate, and the present-day concepts of blood coagulation are the result of brilliant, masterful research on the part of many hematologists. It is possible that clotting factors, and their deficiencies or failures, have been somewhat over-emphasized in the tonsillo-adenoidal problem, and that the majority of such bleeding cases represent a defect in the vascular factors involved; therefore, a study of the mechanism of blood coagulation has been undertaken, with analysis of the hematologic factors involved. The theories of coagulation are diagramed and the various blood tests are discussed in the light of their advantages and disadvantages. The role of the vascular bed, particularly the capillary, is studied, and the prophylactic use of two relatively new compounds in the light of their apparent benefit in prophylaxis of hemorrhage is discussed.

In 1905, Morawitz elaborated the classical theory of blood coagulation:

1. Prothrombin + thromboplastin + calcium ——→ thrombin.
2. Thrombin + fibrinogen ——→ fibrin (clot).

Since then, this theory has been discovered to be inadequate by the many exhaustive and brilliant researches into the study of blood coagulation. Today, blood clotting is known

*Submitted as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, 1957.

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Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 24, 1957.

as the dynamic process in which positive forces are balanced by negative contrary ones, with intensely complicated reactions occurring throughout, both enzymatically and stoichiometrically. The impetus for the new theories was given by Howell in 1910, and now recognized as three distinct stages, or phases, in the coagulation of the blood: *1.* The activation of thromboplastin (the thromboplastin generation); *2.* the formation of thrombin, an enzyme—not occurring naturally in the blood, and *3.* the production of fibrin. The dynamics of the process should be emphasized for, as Alexander describes it; "Dormant and yet delicately poised in the circulating blood, the process is triggered, then assuming ever-increasing velocity wherever a break in vascular integrity occurs, until the final goal of hemostasis is achieved. The importance of emphasizing dynamics, rather than quantity, is clearly illustrated by the fact that, in many hemorrhagic disorders, large and voluminous clots may form, but simply not fast enough."

There is a definite connection between vascular function and coagulation of shed blood in hemostasis, but, unfortunately, there is not as great an understanding of the former. It is estimated that, in approximately 50 per cent of all hemorrhagic diatheses, there is no demonstrable error in coagulation and, in many of these cases, abnormalities of the vascular tree, particularly of the small vessels and capillaries, is the primary cause. Platelets are essential for the integrity of the vessels, which will be mentioned later in greater detail. It is also possible for abnormalities of vascular function and blood coagulation to co-exist, to hinder, or prevent, hemostasis.

A present theory of blood coagulation may be listed in chart form, but the dynamics should always be borne in mind. Following is a diagramatic sketch of one popular theory (Ratnoff):

Phase I.

1. Plasma thromboplastin—
    1. AHF (antihemophilic factor).
    2. PTC (plasma thromboplastin component).

3. PTA (plasma thromboplastin antecedent).
4. Thromboplastin component.
5. Hagerman factor.

2. Platelets—from megakaryocytes.

3. Wettable surface.

↓

Thromboplastin

Phase II.

Prothrombin

Thromboplastin ↓ Vitamin K, CA++, Proconvertin (Factor VII, SPCA), Proaccelerin (Factor V, AC globulin)

Thrombin

Phase III.

Thrombin

↓

Fibrinogen ——→ Fibrin.

Coagulation of the Blood.

From the initial, simple, classical theory of Morawitz, the theories of blood coagulation have become increasingly complex but more accurate. Unfortunately, from different investigators and laboratories using varied techniques, there has arisen a myriad of terms, most of which refer to the same component or substance under different names. Undeniably, the research has been brilliant, but there is need for adoption of a common terminology for purposes of clarification. Below is a table of synonyms after Stefanini and Wintrobe:

1. Platelet thromboplastic factor—

a. Thromboplastinogenase, cellular thromboplastic component (TCC).

2. Plasma thromboplastic factor—

a. Prothrombokinase, plasmokinin, thromboplastinogen, thromboplastic component (TPC, not PTC).
b. AHG, AHF (antihemophilic globulin or factor).

3. Thromboplastin (tissues)—
a. Thrombokinase, thrombokinin.

4. Factor V—
a. Labile component, labile factor.
b. Proaccelerin ——→ accelerin.
c. Plasma AC globulin ——→ serum AC globulin.

5. Factor VII—
a. Stable component, stable factor.
b. Proconvertin ——→ convertin.
c. Plasma precursor ——→ SPCA (serum prothrombin conversion accelerator).

Stefanini lists where the various coagulation factors are found in the blood:

1. Platelets—
a. Platelet thromboplastic factor.
b. Platelet accelerators 1 and 2.
c. Platelet factor 3 (antiheparin factor).

2. Plasma—
a. AHF.
b. PTC.
c. Prothrombin.
d. Calcium.
e. Factor V and VII.
f. Fibrinogen.
g. Anticoagulants.
a. Antithromboplastin, antithrombin, Albumin X (heparin co-factor), profibrinolysin, antifibrinolysin.

3. Serum—
a. All plasma components (except fibrinogen) in proportion to amount utilized in coagulation.
b. Serum AC globulin.
c. SPCA (stable component).
d. Thrombin and metathrombin.

First Phase of Coagulation.

Anti-hemophilic factor (AHF or AHG) is a euglobulin found in the plasma and associated with fibrinogen. Its deficiency causes classical hemophilia. The precise role and fate in coagulation, as well as the site of synthesis is obscure, although it is essential for normal thromboplastic generation, along with plasma thromboplastic component (PTC) and plasma thromboplastic antecedent (PTA). AHF is consumed in coagulation.

Plasma thromboplastic component (PTC) is not consumed in coagulation and is, therefore, present in serum in a concentration compatible with its rate of utilization. Its deficiency causes Christmas disease, similar to hemophilia, and indistinguishable from it by routine blood tests.

Plasma thromboplastic antecedent (PTA) is a globulin, producing in deficiency a disease clinically similar to hemophilia. It is also present in serum.

Possible fourth and fifth components have been claimed (PTF-D) and also "Hagerman" factor, but further studies are needed on these factors.

When there is a deficiency of any one of these factors, prolonged bleeding time and impaired prothrombin consumption are usually found. Clinically, hereditary patterns have permitted differentiation when no other methods have succeeded.

The platelets are formed from the cytoplasm of megakaryocytes in the bone marrow, and are present in circulating blood in normal quantity of 150,000 to 500,000 per cc. They are destroyed in the spleen after utilization or ageing. Theirs is a fundamental activity in coagulation, but the order of activity has brought about disagreement. It is felt that platelets do not initiate coagulation but they do supply an important thromboplastic factor, sometimes termed thromboplastinogenase (Quick), which activates thromboplastinogen (AHF) in plasma. Contact with the injured surface of the blood vessel and/or tissue, plus the action of fibrin upon the platelets in the autocatalytic process of coagulation, causes

lysis and agglutination of the platelets, thus permitting them to function.

There are four basic functions of the platelets with numerous secondary, but nevertheless important, activities. They are essential for the integrity of the vascular tree, particularly the capillaries, which become fragile when the platelets are deficient to the point of 20 per cent of normal. They influence the structure of the fibrin clot by anchoring the fibrin strands, and are an integral part in the process of clot retraction (syneresis). If platelets are below 70,000 clot retraction is diminished or absent. Platelets liberate a vasoconstrictor substance, called thrombotonin by Quick, which locally constricts the affected vessel or vessels. As mentioned, after undergoing lysis, they liberate an activator for the inert circulating AHF. The platelets produce small white thrombi which seal off the capillaries. They also furnish substances that accelerate prothrombin and fibrinogen reactions to form thrombin and fibrin, respectively. The platelets also furnish an anti-heparin factor.

A deficiency in platelets produces thrombocytopenia purpura. A blood smear is important in revealing the numbers of platelets, but not their physiological activity. There is a clinical entity termed "thromboasthenia," in which there are a normal number of platelets, but their function is inadequate. This may be an early stage of thrombocytopenia purpura. One finds a prolonged bleeding time, increased capillary fragility, poor clot retraction and poor prothrombin consumption in this disease, plus a low platelet count on blood smear.

Thromboplastin is largely produced from the cephalin-rich intracellular tissues of the body upon injury as a lipoprotein. It is also formed in the circulating blood by the interaction of the platelet factor, AHF and PTC in the presence of calcium.

Second Phase of Coagulation.

The greatest advance in the study of blood coagulation in the last decade has been in the prothrombin conversion phase. Prothrombin is synthetized in the liver, and Vitamin K is

essential for its production. In the normal adult, there are 300 units of prothrombin per cc. of plasma. There is also a small or variable amount in the serum. It has been demonstrated that if prothrombin is less than 30 per cent of normal, there is a tendency toward hemorrhage, although Quick believes this to be closer to 20 per cent. Vitamin K is present in leafy vegetables and in intestinal bacteria, and naturally occurring Vitamin K is better for replacement therapy than are the synthetic compounds. Vitamin K requires bile salts for its absorption from the GI tract. In severe liver disease, the formation of prothrombin is seriously affected, more so than is fibrinogen, also formed in the liver. There are two accessory factors of prothrombin essential for the clotting mechanism, Factor V and Factor VII. Factor V is also called proaccelerin, AC globulin and labile factor. Factor VII is termed the stable factor, proconvertin and SPCA (serum prothrombin conversion accelerator).

Proaccelerin, or Factor V, is a precursor activated to produce accelerin, which is an active principle governing the velocity of prothrombin activation. Proconvertin, Factor VII, is a prerequisite for thrombin formation under physiologic conditions. Proconvertin and thromboplastin first interact in the presence of calcium to produce convertin. This is the first and foremost prothrombin-converting principle formed during normal clotting. Without it no thrombin is formed under physiologic conditions. This, therefore, is the primary reaction, whereas the proaccelerin-accelerin principle is a secondary reaction which greatly accelerates the velocity of the reaction.

Factor V is a thermolabile protein converted by the action of thrombin to the much more active serum accelerator globulin (accelerin), which is very labile. Prothrombin and Factor V act in conjunction with one another, but it has not been decided whether enzymatically or stoichiometrically. If Factor V is deficient, prothrombin conversion is retarded, producing an increased clotting and prothrombin time, decreased or incomplete prothrombin consumption and decreased thrombin. If it is absent there is virtually no formation of thrombin.

Factor VII is believed by Seegers to be an altered form of prothrombin. It is a relatively stable substance and forms convertin by interacting with thromboplastin in the presence of calcium. It acts in an obscure way upon prothrombin, probably by way of the platelets or thromboplastin. A deficiency will produce retarded thrombin formation.

In Vitamin K deficiency and obstructive jaundice there is a decreased amount of prothrombin but not Factor V, whereas in severe parenchymatous liver disease there is deficiency of prothrombin and both Factors V and VII. Clinically, Factor V (proaccelerin) is rarely deficient. Hypoprothrombinemia may be congenital or acquired. The level of prothrombin may be determined by the prothrombin time, one or two stage test, which can be supplemented by the prothrombin consumption test.

The role of calcium is least understood of the factors. It is essential that calcium be in the bound estate for prothrombin conversion. Rarely is there a deficiency of calcium of such a degree as to interfere with blood coagulation. The normal value of calcium is 6 mg. per 100 cc. of blood.

Third Phase of Coagulation.

Fibrinogen has the greatest molecular weight of all the plasma proteins, and is the most labile. It is synthetized in the liver and thus parenchymatous disease may cause a deficiency state, although not as readily as prothrombin. Congenital afibrinogenemia is well-known, although not common. Fibrinogen is never found in the serum. The normal level is 300 mg. per 100 cc. of plasma. The critical level for disturbance in clotting is 100 mg. per 100 cc. of plasma. A normal prothrombin time excludes a deficiency state of fibrinogen as a cause of hemorrhage, and the coagulation time is normal until less than 30 mg. per 100 cc., therefore being of little practical value in the diagnosis of fibrinogen deficiency.

The fibrin clot is formed by the formation of a fine network of fibers that entangle the formed elements of the blood. This is brought about by the breakdown of fibrinogen to

form needle-shaped fibrils aligning themselves into fibers. These are elastic with the normal clot retracting to 40 per cent of its original volume, largely due to the action of the intact platelets. Fibrin also acts on platelets to cause agglutination and lysis, according to Owren. Fibrin also adsorbs thrombin on its surface, thus aiding in the cessation of the clotting reaction. Some authorities consider this the most important mechanism.

Natural Inhibitors of Coagulation.

Fibrin is a physiologic antithrombin, in that it adsorbs thrombin on its surface. This is a very important reaction in ridding the blood of circulating thrombin. This is called antithrombin I by Seegers. Antithrombin II is considered the plasma cofactor of heparin (albumin X). Antithrombin III acts directly upon the thrombin, neutralizing its activity. It is found in both the plasma and serum. Antithrombin IV is not so well understood, but is found in the plasma and interferes with the prothrombin activation mechanism.

Heparin is found in the granules of the mast cell, which lie among arterioles, venules and capillaries for the most part. Heparin is a mucopolysaccharide. It slows the rate of interaction of thrombin and fibrinogen. It hastens the adsorption of thrombin on fibrin. Heparin also has an antithromboplastic effect. True cases of hyperheparinemia are rare, and those reported have been disputed by many authorities.

Antithromboplastin is a lipid and is thought to block the reaction between tissue, thromboplastin, and Factor VII (proconvertin).

In the globulin fraction of the plasma, a precursor substance, plasminogen, exists which is activated to form plasmin, a fibrinolytic enzyme. This aids in the dissolution of the fibrin clot, assisted by proteolytic enzymes derived from the tissues and by leukocytic action. Platelets, however, contain a powerful antifibrinolysin (Seegers).

It is now possible, in the light of the foregoing, to exemplify the mechanism involved in blood coagulation. Coagula-

tion is initiated by the contact of the blood with an injured surface, thus leading to a series of reactions commonly listed as stages, or phases.

Stage 1—Formation of thromboplastin in the blood, which occurs relatively slowly, and liberation of thromboplastin directly from the tissues. This is the phase of thromboplastin generation.

Stage 2—Active thromboplastin reacts with several factors (calcium, Factors V and VII) to convert prothrombin into thrombin. This is an autocatalytic or chain reaction, which after some thrombin is formed, proceeds with ever-increasing velocity until thrombin evolution is explosive. This is the prothrombin conversion phase.

Stage 3—Fibrinogen is converted into fibrin by the enzymatic action of thrombin, and a clot is formed. A fibrinopeptide is split off. Subsequent steps involve neutralization, or destruction of the excess thrombin plus solidification and retraction of the clot.

AHG and Factor V are first consumed during normal clotting and then prothrombin, therefore leading some authorities to believe their reactions are stoichiometric. PTC, PTA and Factor VII are not consumed and, therefore, are considered to act as enzymes.

THEORIES OF COAGULATION.

It is possible to include diagrams of the five theories of blood coagulation in a cyclic manner: (Owren, Quick, Seegers, Tocantins and Stefanini). Following are the five theories, and Owren's will be described in detail.

Theory of Owren.

1. Tissue injury yields thromboplastin directly, while contact causes disintegration of platelets and release of a platelet factor which, together with AHF and other factors in the presence of contact and calcium, produces thromboplastin.

2. Thromboplastin and proconvertin in the presence of calcium form convertin.

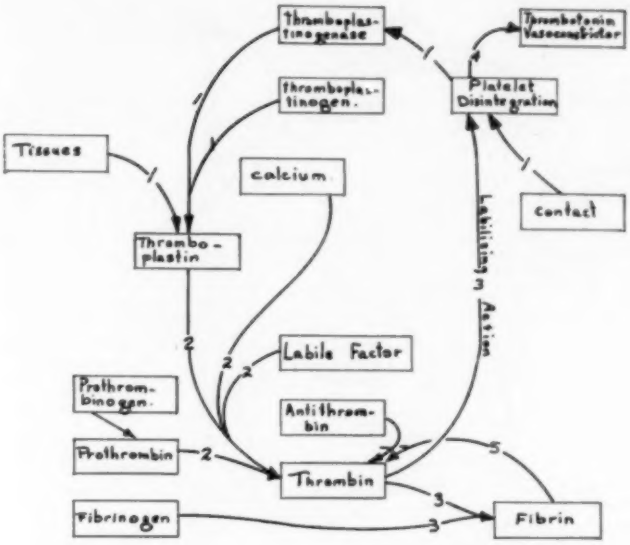


Fig. 1.

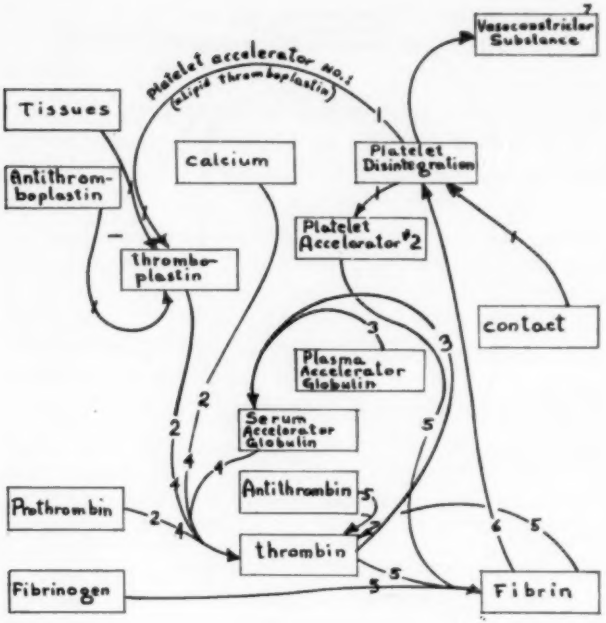
3. Convertin and calcium produce minimal conversion of prothrombin into thrombin.

4. This initially formed thrombin starts the accelerator system, that is, the conversion of proaccelerin to accelerin.

5. Accelerin accelerates the conversion of prothrombin into thrombin in the presence of convertin and calcium.

6. Thrombin is now in sufficient quantity to convert fibrinogen to fibrin.

7. Fibrin provokes further disintegration with further release of thromboplastic substance already mentioned.



theory of L.M. Tocantins 1951

Fig. 2.

8. Natural inhibitors at all times during the reaction are in force.

It is thus possible to sympathize with Stefanini when he writes, "In the field of blood coagulation, there is a crying need for a unified terminology and a critical evaluation of all theories in the light of established facts."

While there are similarities and dissimilarities, there is still considerable agreement. The disagreement occurs chief-

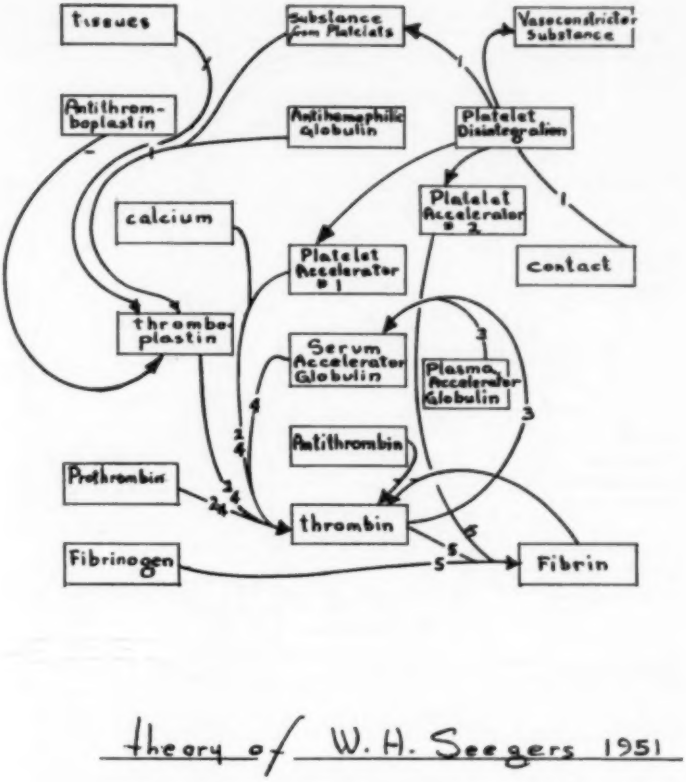


Fig. 2.

ly in the study of the prothrombin conversion phase, wherein Quick, for example, does not favor any accelerator system and feels the reactions are stoichiometric; Seegers and Tocantins favor one accelerator system, while Owren believes in one accelerator plus one converter system; however, all authorities seem to agree that the agglutination or disintegration of the platelets is an initial step which activates thromboplastin and releases the vasoconstrictor principle. They all believe also that one or two accessory factors are essential for prothrombin conversion besides calcium and

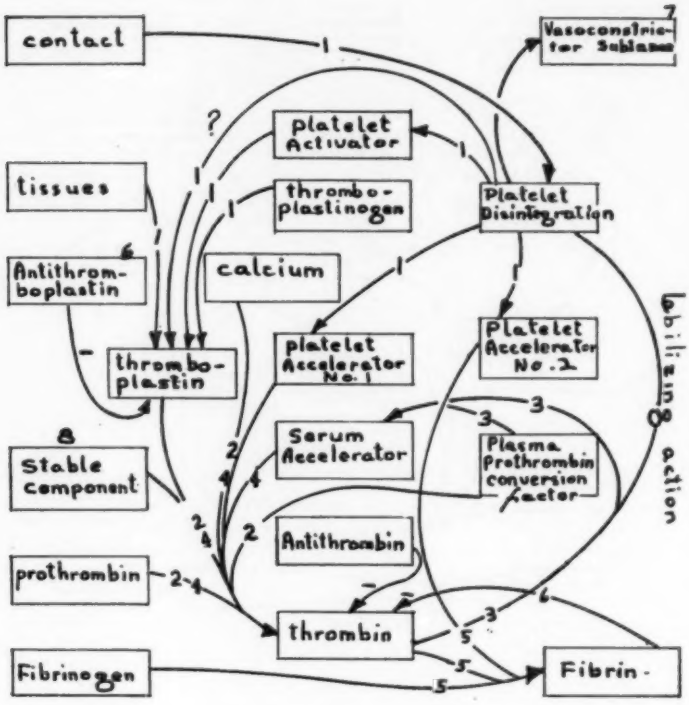


Fig. 4.

thromboplastin. They feel that fibrin plays an active role in coagulation by adsorbing thrombin and labilizing the platelets. It is agreed that positive and negative forces abound, and that, with the positive factors, there are also inhibitors of blood coagulation. Finally, all authorities concur in the

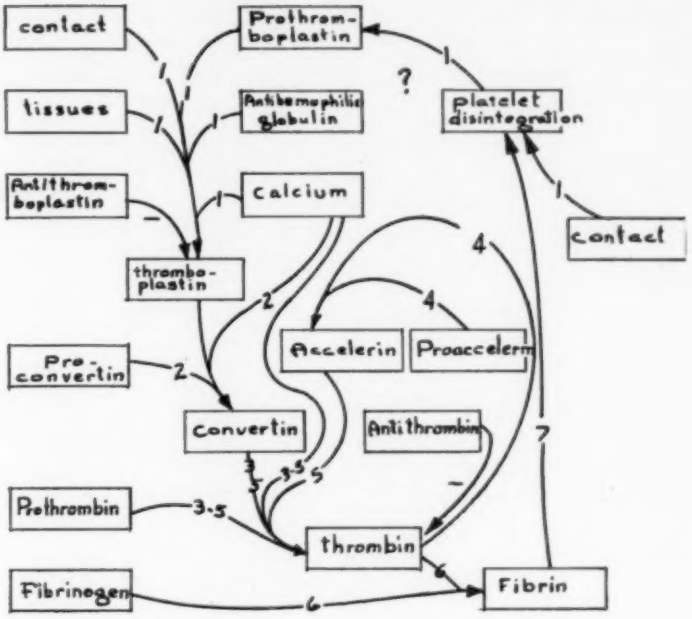


Fig. 5.

autocatalytic, or chain reaction, mechanism in the process of blood coagulation.

VASCULAR FACTORS IN HEMOSTASIS.

Tocantins says the "spontaneous arrest of bleeding involves more factors than those contributed by the blood alone." Spaet agrees when he states "there is an intimate relationship between the integrity of the blood coagulation

system and the integrity of the blood vessel wall." Alexander likewise emphasizes the importance of this relationship, as does Seegers. All authorities admit, however, that not enough is known concerning the role of the vascular bed in hemorrhage.

Concerning the vascular tree, the location of the vessel in many ways affects bleeding. For example, a superficial vessel bleeds more readily because there is little or no tissue contraction. Thus the tissue tension or elasticity is important. The structure of the vessel is vital, in that healthy vessels have elasticity due to their muscle and elastic fibers and are capable of distention and contraction. The reflex motor pathways also affect contraction and dilatation.

Tocantins explains the dynamics of hemostasis as a result of two factors: *1. Vascular*—Pressure in the vessel drops; it collapses, and there is increased surrounding tissue tension, due to escaping blood. *2. Hematogenous*—The blood slows and collects at the site of injury, and the autocatalytic reaction begins. This reaction, he points out, varies according to the type and caliber and location of the vessel and, in relation to the volume, pressure and other properties of the shed blood.

There is a massing of platelets at the site of injury, some of which swell and burst and thus enter the blood coagulation cycle. The remainder shrink and adhere to the surface, to which fibrin may be deposited and other platelets clump. There is later contraction and hardening of the platelet-fibrin plug. It is thought that contraction of the clot is effective in pulling the vessel walls together and reducing the size of the vessel wall and, therefore, the greater the ability of the clot to become tough and hard, the greater is its ability to withstand the rise in blood pressure and subsequent reflex dilatation of the blood vessel.

Capillaries consist of loops linking arterioles and venules. The pressure at the arteriolar end is 32 mm. of mercury and at the venule end, 12 mm. There are 16-64 capillary loops per sq. mm. of skin surface. There are more platelets in the arterioles than venules. The capillary wall consists

of a fibrillary basement membrane, endothelial cells which can swell with stimulation and intercellular cement. The mechanism of clotting in the capillaries is thought of as occurring in the following manner: An initial injury occurs, which produces a transient vasoconstriction. A platelet plug is formed, followed by a fibrin clot in the manner heretofore described. There is then a secondary vasoconstriction action produced by the liberation of a specific vasoconstrictor substance from the platelets (thrombotonin). "The rate of clotting is less important to hemostatic efficiency than is usually supposed, and other attributes of the clots, such as toughness and retractility, may have a greater influence" (MacFarlane).

It has been shown that the capillaries contract actively and do not undergo a passive collapse. This is brought about by the motor elements, wherein a sympathetic nervous system stimulates the stellate cells which reinforce the capillary walls. There is also a direct primary vasoconstricting action of circulating and tissue substances, as well as a secondary action of platelet-released thrombotonin. The capillaries can contract against a pressure of 60 mm. of mercury or less. Once contracted, they can resist a pressure of 100-110 mm. of mercury without redilating. Capillary dilatation can result passively from increased systolic pressure (over 110 mm. of mercury) or actively, due to the liberation by the forces of trauma, heat or cold, or of a histamine-like (H) substance from the nerve endings; therefore, the method of capillary hemostasis is thought to be the following: An injury produces hemorrhage, and the capillaries are dilated by the "H" substance liberated from the injured tissues. The "H" substance is removed by blood flow or by diffusion of fluid. The capillaries then contract and bleeding ceases with the assistance of agglutinated platelets acting as a plug. The fibrin clot is formed by the autocatalytic reaction. The clot then retracts and becomes tough and hard and firmly attached to the vessel. The capillary redilates, but the firm blood clot acts as a plug and maintains hemostasis.

Failure of hemostasis is, therefore, due to loss of contrac-

tility of the capillary, or defective blood coagulation, or a combination of both. In defective blood coagulation, in which a firm clot is not formed during the period of capillary contraction, when the blood vessel dilates, the clot cannot withstand the increased pressure, and hemorrhage results.

LABORATORY TESTS.

There are numerous blood tests that are used in the study of hemorrhagic states, in routine preparation of the patient for surgery, or in a routine physical examination. It would be well to analyze these tests in order to determine their fitness to aid in diagnosis of a hemorrhagic defect or a vascular disorder, or both. Alexander recommends that simple procedures be employed first such as, *1.* the capillary fragility test; *2.* a routine CBC with careful scrutiny of the stained blood smear; *3.* the bleeding time (Duke or Ivy method); *4.* the clotting time (Lee-White); *5.* clot retraction and lysis; *6.* the one-stage prothrombin time; *7.* the prothrombin consumption test. If all these tests are within normal limits, the hemorrhage is more likely due to a vascular disorder.

SPECIFIC LABORATORY TESTS.

I. *Coagulation Test.*

The coagulation, or clotting time, is a measure of the intrinsic power of the blood to convert fibrinogen to fibrin. It must be performed on venous blood, and the most universally accepted method is that of Lee-White. The normal value is 4-6 minutes. It is not considered a sensitive test of hemorrhagic disorders, unless there is a severe alteration of the clotting mechanism. Its most practical use is in the control of heparin and dicumarol therapy. An increased value is always significant, but a normal result does not exclude a hemorrhagic disorder. The coagulation time may be prolonged, but not necessarily unless the condition is severe, in hemophilia, hypoprothrombinemia, afibrinogenemia and hyperheparinemia (rare).

II. *The Bleeding Time.*

The bleeding time is the time required for bleeding to stop after a stab wound in the skin which is not greater than 4 mm. deep. There are two commonly accepted methods: Duke and Ivy. The normal values are 1 to 3 minutes and 1 to 4 minutes, respectively. Slightly prolonged is 4 to 5 minutes and prolonged is greater than 5 minutes. It is not a measure of the capillary fragility or hyperpermeability, but rather the response of the capillaries to injury. It is prolonged in thrombocytopenia purpura, severe liver disease and severe deficiency states of the various clotting factors. Surprisingly enough, the bleeding time is not affected by scurvy nor hereditary hemorrhagic telangectasia.

III. *Platelet Count.*

The platelet count does not reflect bone marrow production, nor does it reveal the state of the physiological activity of the platelets. Normal values are considered between 150,000 and 250,000 per cu. mm., although Quick believes 500,000 per cu. mm. the upper limit of normal. Platelets are deficient in thrombocytopenia purpura of primary or secondary origin.

IV. *Clot Retraction.*

This measures the spontaneous shrinking of the blood clot, with the expression of clear serum. The degree and speed of the clot retraction depends chiefly upon the numbers of platelets and the quantity of thrombin. An increase of these substances will speed the contraction. On the other hand, an excess of fibrinogen decreases it, and the greater the cell volume the less the contraction; however, this test is an indirect measure of the platelet count and the normal reaction is one in which retraction occurs in 30 to 60 minutes.

V. *Capillary Fragility (Rumple-Leed, Tourniquet Test).*

This test consists of obstructing the venous flow and measuring the number of petechiae formed in a unit area distal to the obstruction. By raising the intracapillary pressure it discloses a latent hyperpermeability, and the number and

size of petechiae is roughly proportional to the bleeding tendency.

Interpretation is difficult when positive results are obtained (more than five petechiae). It is positive in a great variety of conditions, but it is not a factor in many hemorrhagic problems. Its relationship to the bleeding time and the platelet count is inconsistent and, therefore, is of limited value in the study of the hemorrhagic disorders. Capillary fragility is increased under a number of circumstances in which there is no defect in blood coagulation, infectious diseases and chronic nephritis among others, for example.

VI. *The Prothrombin Time.*

This is the time needed for the formation of a clot by adding thromboplastin reagent and calcium choride to oxalated plasma. The time is a measure of the prothrombin level of the blood, since the concentration of thrombin is determined by the speed of coagulation. The hemorrhagic tendency is considered critical at 20 seconds and above. A time of 25 seconds indicates a prothrombin level about 20 per cent of normal.

The test most universally used is the one-stage method of Quick; but it has been modified many times, so that in many laboratories a one and two-stage prothrombin time is now used. This test is considered a very reliable method of determining prothrombin quantity, but actually prothrombin activity is measured.

VII. *Prothrombin Consumption Test.*

This test is the quantative determination of the prothrombin remaining in the serum after the blood has coagulated under standardized conditions. In hemorrhagic disease, the residual serum prothrombin is increased after coagulation is completed. If there is less than 15 per cent serum prothrombin, as compared with the parent plasma prothrombin before coagulation, the test is considered normal. "It is an extremely useful diagnostic procedure whereby a normal value readily excludes many serious hemorrhagic diseases" (Alexander).

There are also various other tests which can be employed, but usually not routinely nor in the average laboratory:

1. Plasma Clotting Time. This measures more pronounced deficiency states of AHG, PTC and PTA, and is a fairly simple test to perform.

2. Thromboplastin Generation Test. It is a useful method for determining impaired formation of blood thromboplastin and altered platelet function. It is useful in determining mild AHG, PTC and PTA deficiencies.

3. Circulating Anticoagulant Test. This is a laborious, but sensitive, test in which a circulating anticoagulant can be detected. More clinical cases of circulating anticoagulant are being discovered by these tests.

4. Fibrinogen Test. This is based on the conversion of fibrinogen to fibrin and the amount of fibrin is measured.

Alexander lists the following interpretations of a routine laboratory test: An increased clotting time indicates retarded coagulation, but a normal test does not rule out a hemorrhagic defect. A normal prothrombin time excludes deficiency of prothrombin, AC globulin, or Factor V, Factor VII and fibrinogen. If the prothrombin consumption is poor, but the prothrombin time is normal, the abnormality lies in the first phase of clotting, the thromboplastin generation; thus, more complicated tests are needed for the measurement of AHF, PTC, PTA, platelet function, antithromboplastins and others. A second phase defect is readily revealed by an increased prothrombin time; however, further tests which are not readily available are necessary to determine which factor, or combination of factors, is involved. The prothrombin time is increased also in the third stage of coagulation, if a disorder exists.

Quick feels that, in most hemorrhagic diseases, only three tests are required for diagnosis: the bleeding time, prothrombin time and the prothrombin consumption test.

Wintrobe lists the causes of alterations in the various

measures of coagulation. His chart has been somewhat altered.

Laboratory Finding	Condition
I. Prolonged coagulation time.	1. Hemophilia (decreased AHG). 2. Decreased PTC. 3. Decreased PTA. 4. Hyperheparinemia (rare). 5. Excess circulating anticoagulants. 6. Hypo- and afibrinogenemia (congenital or acquired). 7. Hypoprothrombinemia (less than 20% of normal). a. Deficiency of prothrombin, Factor V or VII.
II. Increased prothrombin time (one stage).	1. Increased dicumarol (causes deficiency of prothrombin and Factor VII). 2. Parahemophilia (decreased Factor V). 3. Decreased SPCA (Factor VII deficiency). 4. Vitamin K deficiency. 5. Liver disease (impaired production of prothrombin, Factor V and VII).
III. Prothrombin consumption reduced.	1. AHG, PTC, PTA decreased (impaired thromboplastin production). 2. Thrombocytopenia (impaired thromboplastin production). 3. Thromboasthenia (impaired thromboplastin production). 4. Circulating antithromboplastin (impaired thromboplastin production).
IV. Prolonged bleeding time.	1. Thrombocytopenia purpura (platelet deficiency. 2. Any severe cause found in Items I and II. 3. von Willebrand's disease (abnormality of capillaries of skin and mucous membrane).
V. Positive capillary fragility.	1. Non-thrombocytopenia purpura. 2. Thrombocytopenia purpuras. 3. Scurvy.
VI. Thrombocytopenia.	1. Thrombocytopenia purpuras, primary and secondary.
VII. Poor clot retraction.	1. Thrombocytopenia purpuras. 2. Thromboasthenia.

There are two substances which have received a great deal of interest and enthusiasm recently in the treatment of hemorrhagic problems. They are of value chiefly in capillary disorders, but seem to be of no value in faulty coagulation of the blood. They appear to be ideal in the management of the tonsillectomy and adenoidectomy states, and the preliminary work that has been done indicates that they will prove to be useful compounds in the management of this

surgery. It is felt that the majority of complications following tonsillectomy and adenoidectomy is due to the failure of normal healing reaction at the site of the tonsil and adenoid bed. Since these compounds are known to aid the capillaries, in that they strengthen the capillary wall, tend to restore capillary permeability to a normal level, decrease capillary fragility and increase capillary resistance, authorities are of the opinion that these drugs have a definite use in the treatment of the tonsillo-adenoidal patient. They are called the flavonoids, or bioflavonoids, and adrenochromazone.

THE FLAVONOIDS.

The flavonoids (flavones, flavonones and flavonols) are unsaturated ketones which occur naturally in plants and vegetable dyes and produce their ivory or yellow coloring. They are usually present as glycosides in the sap. The group of compounds is usually referred to as Vitamin P, which is distinct from Vitamin C, and its best-known components are hesperidin, eriodictyol glyoside (demethylated hesperidin), quercetin and quercitrin. Rutin is considered a flavonoid, is a rhamnoglycoside, having an action similar to Vitamin P, and is extracted from lemon rind. The term bioflavonoid refers to those substances which possess biological activity.

The flavonoid compounds, according to Beiler, have a "function in regulation of permeability and maintenance of capillary integrity." "These compounds," he continues, "are capable of interacting with various metabolites and enzyme systems."

Most authorities attempt to differentiate between capillary permeability (loss of protein) and capillary fragility (escape of erythrocytes), and it is felt that the flavonoids primarily affect fragility rather than permeability. In practice, however, it appears difficult to separate these two factors of capillary physiology or pathology.

The capillary wall consists of a parchment-like endothelial lining with intercellular cement, a non-cellular endocapillary lining and a pericapillary sheath. The endothelial lining and intercellular cement substance are the most important con-

stituents, and it is these structures that are affected by the flavonoids acting synergistically with Vitamin C. It is important to emphasize the relationship with Vitamin C, because most of the flavonoid physiological activity depends upon the presence of Vitamin C. The latter also requires Vitamin P for certain of its functions.

The flavonoids and Vitamin C are known to act directly upon the capillary wall to strengthen the endothelial lining and its intercellular cement, although the exact mechanism of action has not been demonstrated. Vitamin P potentiates the action of ascorbic acid. It serves as an inhibitor of hyaluronidase and of histamine. It also acts in a non-specific manner by virtue of its antioxidant properties to prevent the oxidation of epinephrine, and Schiller believes the flavonoids have a strong independent vasoconstriction of their own. Vitamin P also appears to have a direct effect upon the bleeding and coagulation time, although this has not been made clear to most investigators.

The flavonoids have been used clinically in a variety of conditions in which there appears to be increased capillary fragility and thus lowered capillary resistance:

1. Vascular purpura.
2. Habitual abortion.
3. Eczema and psoriasis.
4. Diabetes and diabetic retinopathy.
5. Hematuria and hemorrhagic nephritis due to infection, allergy or drug reactions.
6. Hypertension.
7. Gastrointestinal hemorrhage.
8. Postirradiation hemorrhagic tendencies.

In diabetes, diabetic retinopathy and hypertension, the majority of authorities feel the flavonoids are ineffective, but in the other conditions, the results appear beneficial.

Goldman recently has advocated the prophylactic use of Vitamin P in association with Vitamin C in tonsillectomy and adenoidectomy cases. It is his impression that in his 530 cases there was much less initial hemorrhage, less oozing

in the immediate postoperative period, and less late secondary hemorrhage. Unfortunately, this is an impression and no actual laboratory data is offered as proof of the effectiveness of the compounds. He does make the important observation, however, that the flavonoids and Vitamin C are more effective when given prior to surgical trauma, than after hemorrhage has commenced postoperatively.

There is a commercial preparation, called "CVP," which is produced in either capsule or syrup form. Each capsule or each 5 cc. of the syrup contains 100 mg. of citrus bioflavonoid compound and 100 mg. of ascorbic acid. There is also a preparation available with Vitamin K, which provides in each tablet or 5 cc., 0.66 mg. of Menadione, in addition to the 100 mg. of citrus bioflavonoid compound and 100 mg. of ascorbic acid. Goldman suggests that younger children (two to four years) be given 5 cc. of the syrup three times daily for six to eight days prior to surgery and for four days postoperatively, with the dosage doubled for older children. For adults, he recommends two capsules four times daily over the same period of time. If a prothrombin deficiency is suspected, it is possible to administer the drug with Vitamin K added.

Martin states that the "anatomical and physiological importance of the capillary is directly correlated with the biochemical basis of action of the bioflavonoids and, in turn, with that of ascorbic acid." He also states that in cases of diseased or failing capillaries, the flavonoids and ascorbic acid are indicated as the therapeutic substances.

ADRENOCHROMAZONE.

Adrenochrome is a systemic hemostat, unstable, being destroyed within four hours. Its monoxime and semi-carbazone forms are stable, but insoluble. When linked with sodium salicylate complex, the resulting compound is both stable and soluble and capable of clinical application. It is termed adrenochrome monosemicarbazone sodium salicylate complex. The salicylate radical is too small to be of significance in hemorrhage.

Adrenochrome is an oxidation product of epinephrine, but

has no sympathomimetic properties. The adrenochromazone complex will not affect blood pressure, cardiac rate nor volume; it will not interfere with blood chemistry nor its coagulation. Vitamin K, anticoagulants and coagulants are likewise not affected, and they may be used in conjunction with adrenochromazone with impunity. The antihistamines, however, retard its action and should be discontinued for 48 hours before its use. The complex is excreted through the kidneys within 12 hours, but there is a residual capillary effect for approximately 48 hours.

Adrenochromazone has a high safety therapeutic level and is non-cumulative in the patient. Since it has no sympathomimetic properties, and since no toxic effects have been demonstrated, there are no apparent contraindications to its use. The mode of action is not known with certainty, but it is apparent that the complex acts upon the capillary endothelial cellular layer and perhaps the intercellular cement to reduce capillary permeability and to increase capillary resistance. The drug will not affect the larger vessels, nor arterioles, due to its lack of sympathomimetic action. Some observers feel, however, that adrenochromazone promotes retraction of severed capillary ends, but again the mechanism is not certain.

The commercial product, termed "Adrenosem," is supplied in ampules of 1 cc. of 5 mg. strength, tablets of 1 and 2.5 mg., and syrup 2.5 mg. per 5 cc. It is a hypertonic solution and must, therefore, be administered intramuscularly, and not intravenously. There is a transient stinging sensation which is unpleasant. The oral dose is thought to be one-third as effective as the parenteral dose due to the rapid oxidation of the complex in the gastro-intestinal tract. Sherber regards a dose of less than 5 mg., either orally or parenterally, as of doubtful value.

The use of adrenochromazone has been reported in a variety of cases, such as idiopathic purpura, retinal hemorrhage, familial telangectasia, epistaxis, pulmonary hemorrhage, post-irradiation petechial hemorrhages, uterine bleeding, and post-surgical hemorrhage. It has been reported useful in all

such cases, with dramatic results in some. Bacala reports reduced postoperative tonsillectomy hemorrhage from 19.8 per cent to 7 per cent in an analysis of 1,015 cases, but fails to mention how many adenoidectomies were involved, the age of the patient (children vs. adults), and the method of anesthesia employed (general vs. local). Ownings reduced his hemorrhagic level from approximately 10 per cent to 1 per cent in 102 cases with the use of adrenochromazone. Peele maintained his hemorrhagic level at 3.9 per cent with the use of adrenochromazone in 178 cases, but it was his impression that less sutures were required at the time of surgery, that there was less oozing from the tonsillar and adenoidal areas, and that the time required for the surgery (due to more rapid hemostasis) was reduced. He also reported a significant decrease in hemorrhage in performing submucous resections and when nasal packs were removed.

Sherber concludes that adrenochromazone is a "potent antihemorrhagic factor in conditions in which the integrity of the smaller vessels is interrupted." He advocates the use of the complex in the prevention of vascular accidents incident to hypertension, the maintenance of small vessel integrity, the preoperative use when oozing is anticipated, such as in a tonsillectomy and adenoidectomy, or a prostatectomy, and as an adjunct to the therapy of hemorrhage incident to surgery.

In our tonsillectomy and adenoidectomy cases, as well as nasal operations, we are now employing a dosage of 5 mg. given intramuscularly one hour preoperatively, 5 mg. intramuscularly one hour and again 24 hours postoperatively. Occasionally, we supplement this with oral doses ranging from 5 to 10 mg. once or twice daily for approximately seven days postoperatively, depending upon the nature of the surgery and the patient's hemorrhagic status.

DISCUSSION.

In cases of tonsillo-adenoidal hemorrhage, perhaps too much emphasis has been placed upon the mechanics of blood coagulation itself, and too little upon the vascular factors

involved. There is, of course, a reason for this in that blood coagulation studies can be more easily carried out since shed blood is readily available, whereas tissue analysis is more difficult. Second, the importance of vascular factors in hemostasis has only recently been given necessary recognition, and even now is frequently overlooked. Third, analysis of blood coagulation and the various blood tests have occupied the time and efforts of hematologists for many years and are, indeed, most intriguing scientific pursuits; however, in the tonsillo-adenoidal hemorrhage problem, it is usually true that bleeding is a result of vascular dysfunction, rather than a coagulation defect.

The management of the problem is three-fold: *1.* preoperative analysis of the blood to discover a possible faulty mechanism of blood coagulation; *2.* strengthening of the involved vascular tissues, so that the opportunity for operative or postoperative hemorrhage is reduced; and, *3.* the management of tonsillo-adenoidal hemorrhage, should it occur.

1. Preoperative Blood Analysis. The usual routine preoperative blood studies in most institutions consist of a complete blood count, including hemoglobin, hematocrit, red blood and white blood counts, a differential, and sometimes a platelet count. In addition, bleeding and clotting times are usually performed. This method of study, while helpful, does not reveal all coagulation defects and should be supplemented by further tests. A battery of blood tests is recommended, as mentioned previously:

1. Complete blood count and platelet count.
2. Bleeding time.
3. Coagulation time.
4. Capillary fragility test.
5. Clot retraction.
6. Prothrombin time.
7. Prothrombin consumption test (optional and not available in most institutions).

If these tests are within normal limits, virtually any clotting disorder can be ruled out; should bleeding occur, a

vascular factor is likely responsible (assuming the surgery to be non-contributory).

2. Vascular Factors. The majority of hemorrhages that occur following tonsillo-adenoidal surgery is due to disorder within the vascular system, owing to the trauma of the surgery and the consequent inability of the tissues primarily to aid in coagulation or, secondarily, to heal in a proper manner. Unfortunately, there is no test, or group of tests, which would indicate this possibility, unless the capillary fragility test is abnormal, but this is not necessarily diagnostic of a vascular disorder; however, examination of the patient should reveal poor nutrition which weakens capillaries, and a history of ease of bruising and/or hemorrhage will disclose a possible vascular defect if the preoperative blood tests, as described previously, are normal. The state of nutrition, if poor, should be adjusted to normal before surgery is undertaken. The use of large amounts of salicylates pre- and postoperatively should be discouraged, for it is known they weaken the capillaries, particularly the endothelial lining and the intercellular cement. Flavonoid compounds appear to be useful pre- and postoperatively, not only in patients with a possible vascular disorder, but also in the normal, apparently healthy, individual submitting to this type of surgery. The addition of Vitamin K is necessary only if the prothrombin tests reveal decreased prothrombin. The use of adrenochromazone is likewise recommended in the immediate pre- and postoperaitve period.

3. Management of Tonsillo-Adenoidal Hemorrhage. Several observations can be made on this problem. In all cases of hemorrhage, whether primary or secondary, the usual methods employed should be followed. It is not the purpose of this paper to alter standard, accepted methods of treatment, but rather to analyze the cause of hemorrhage and to prevent it, if possible.

By employing the flavonoids and adrenochromazone and by avoiding the use of salicylates, it is felt the vascular bed of the tonsils and adenoids is strengthened and there is, therefore, less likelihood of hemorrhage. Unfortunately,

these compounds exert their chief effect upon the capillaries, and should the bleeding arise from a venule or arteriole, they are not likely to be effective, either prophylactically or therapeutically. Adrenochromazone, however, appears to be of benefit in the active management of capillary bleeding in these cases, as well as exerting a prophylactic influence, and may, therefore, be employed along with the other usual measures when tonsillo-adenoidal hemorrhage occurs.

Frequently, Vitamin K, in synthetic form, is employed in tonsillo-adenoidal hemorrhage. If the prothrombin level of the patient is normal, as shown by preoperative tests, Vitamin K is not needed and should, therefore, not be used.

CONCLUSIONS.

The routine blood tests that are performed preoperatively are usually inadequate and may, therefore, fail to disclose a hemorrhagic tendency. A complete battery of tests, including bleeding and coagulation times, complete blood count with platelet analysis, capillary fragility tests, clot retraction, prothrombin time and prothrombin consumption tests, is recommended.

The majority of tonsillo-adenoidal hemorrhages are due to a defect of the vascular bed, rather than to a coagulation defect. The strength of the vessels can be improved by insuring a good nutritional state, avoidance of salicylates, and use of flavonoid compounds and adrenochromazone. The employment of Vitamin K in cases of hemorrhage is of little or no value if the prothrombin level is normal.

It is, therefore, believed that emphasis upon prophylaxis of hemorrhage by adequate preoperative blood tests to disclose coagulation defects, and by use of compounds to strengthen the vascular elements, is the proper approach to this aspect of the tonsillo-adenoidal problem.

SUMMARY.

An analysis of the modern concept of blood coagulation has been made and the three phases, or stages, of coagulation

have been outlined, together with the various factors involved in each. The five principal theories of coagulation are shown diagramatically and one theory is discussed in detail. The principal blood tests are discussed with their advantages and limitations. The importance of the role of the vascular bed in hemorrhage is pointed out, particularly that of the capillary. Two newer compounds, the flavonoids and adrenochromazone, are mentioned in the prophylaxis of tonsillo-adenoidal hemorrhage. A recommendation is made for the employment of blood tests in addition to those usually performed to detect possible coagulation defects preoperatively. The use of the flavonoids and adrenochromazone is also recommended prophylactically, because they appear to reduce the incidence of tonsillo-adenoidal hemorrhage.

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573 Fisher Bldg.

THE 11TH CONGRESS OF THE INTERNATIONAL ASSOCIATION OF LOGOPEDICS AND PHONIATRICS.

The 11th Congress of the International Association of Logopedics and Phoniatrics will take place in London, August 17-22, 1959. The following official reports will be presented: The Inheritance of Voice and Speech Disorders, Prof. R. Luchsinger, M.D., Zürich; Defects of Articulation, Muriel Morley, B.Sc., F.C.S.T., Newcastle; The Physiology and Pathology of the Soft Palate, Prof. Lucio Croatto, M.D., Padua. The official languages of the Conference will be: English, French, and German.

Those working in the field of Speech and Voice Therapy and all who are interested in this specialty are invited to attend and to submit papers.

If intending to be present, whether submitting a paper or not, please inform the Congress Secretary at the earliest possible date.

Papers are invited on subjects relevant to the three main reports or any other aspect of speech and voice. Only one contribution will be accepted from any one member of the Congress and this must not have been previously published. The Committee reserves the right to select papers. Papers should be restricted to 15 minutes in length; demonstrations to 10 minutes. In special cases 20 minutes may be allowed for a paper of great importance but only if application is made at the time of submitting the title. Should a group of four or more people wish to present a symposium or a prepared discussion on a theme, consideration will be given to allocating up to one hour for such a contribution.

Titles must be received by September, 1958. Summaries must be received not later than November 15th, 1958.

All communications are to be sent to: Peggy Carter, L.C.S.T., 46 Canonbury Square, London, N. 1.

THE INTERNATIONAL CONGRESS ON THE EDUCATIONAL TREATMENT OF DEAFNESS.

The University of Manchester Department of Education of the Deaf, will sponsor The International Congress on the Educational Treatment of Deafness, July 15-23, 1958.

The Minister of Education for England and Wales has decided to recognize the Congress as a Short Course which teachers of the deaf may attend under Grant Regulations.

An interesting program has been arranged.

Demonstrations will be given to illustrate some aspects of the research that is in progress in the audiology unit and audiological laboratories of the Department of Education of the Deaf, and in collaboration with the Department of Otolaryngology, the Public Health Department and School Medical Service of the City of Manchester, and the Ear, Nose and Throat Department of the Royal Manchester Children's Hospital. The subjects of the demonstrations will include procedures for the making of screening tests and assessments of the hearing of babies and young children, the results of guidance to parents to enable them to give home training to their children of pre-school age whose hearing is impaired, and the results of an experimental investigation on problems in architectural acoustics that concern the use of hearing aids in school buildings.

Arrangements are being made to offer accommodation to members of the Congress in University Halls of Residence. The cost of this type of accommodation is expected to be about £1. 2s. od. per day, including bed, breakfast and dinner. The University refectory will be available for meals during each day.

For those members who wish to do so, arrangements will be made to visit audiology clinics and special schools in London, Oxford and other places. In connection with the Congress it is hoped to organize tours to places of interest. The dates proposed for this are July 9th, 10th, and 11th.

For further details write: Prof. A. W. G. Ewing, Department of Education of the Deaf, The University, Manchester 13, England.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

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President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
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Meeting: Palmer House, Chicago, Ill.

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Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40, Pa., U. S. A.
Meeting: Sixth International Congress of Bronchoesophagology, Philadelphia.

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Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P. M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting:

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President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

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THE LARYNGOSCOPE

VOLUME LXVII OCTOBER, 1957 NUMBER 10

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